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16. Abstract

The monograph describes the problems of decompression disorders: the functional disturbances arising in the organism as the result of the formation of gas bubbles in the blood and tissues incident to decompression. The author and his colleagues present experimental data gathered in the course of many years of systematic study of why and how decompression disorders develop in man and animals after a stay under increased pressure or upon transition to a rarefied atmosphere. The author explains the general principles governing the development of decompression disturbances and the role of aeroembolism in their pathogenesis. He establishes the existence in the organism of defense reactions against aeroembolism, as well as the possibility of enhancing the organism's resistance to decompression effects. He presents conclusions of value to deep-sea diving and to the caisson method of construction work under water, as well as experimental data on how to ensure decompression disorder prophylaxis on space flights.

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Foreword

The fact that the present book differs somewhat in character /5
from monographs devoted to individual problems of physiology obliges me to preface it with a brief foreword. I have not striven after complete and detailed systematic explication of the fundamental problems of decompression disorders in the usual, if one may say academic, sense of successively expounding the etiology, pathogenesis and symptomatology (or, as is often said, clinical picture) of decompression disturbances, their anatomicopathologic bases and methods of treatment and prophylaxis. This book was planned along other lines. Its main purpose is to present experimental data obtained by the author and his colleagues in the course of many years of research aimed at elucidating the most vexing physiological questions of decompression disorders, in particular those questions whose solution is necessary in order to place rational and reliable measures for their prophylaxis on a firm scientific footing. Thus, the entire exposition of the material is subject to the logic underlying experimental research aimed at elucidating the physiological principles governing decompression disorders.

The first part of the book is intended to introduce the reader to the heart of the matter of decompression disorders and focuses attention on the physiological aspects of this problem, which are the most important ones from the scientific and practical points of view.

The second part discusses the results of experimental analysis of the physiological principles governing decompression disturbances in animals on the example of experiments with decompression effects connected with a precedent increased pressure effect.

The third and final part of the book discusses the results obtained mostly with people but partially with animals in experiments aimed at solving an acute problem of the day: placing on a firm scientific footing prophylactic measures against decompression /6
disorders in the case of suited astronauts stepping out of a spacecraft cabin into empty space.

The experimental data presented in this book do not of course pretend to solve these complicated problems, but both the author and the colleagues with whom he shared this experimental labor do hope that it will in some measure bring us closer to elucidation of those physiological questions an answer to which is absolutely necessary if we are to place prophylactic measures against decompression disturbances on a firm scientific footing and safeguard man under those specific conditions of activity that are connected with decompression effects.

I take advantage of this opportunity to express my sincere gratitude to the fellow workers with whom I shared the labor

involved in the experimental part of this study. To A. P. Brestkin I owe a special debt of gratitude for inspiring me to work in this field.

I am especially indebted to my teacher, M. P. Brestkin, under whose guidance the bulk of the experimental and analytical part of the research described in this book was carried out, as well as to O. G.Gazenko, under whose much-esteemed guidance our team is carrying on its work involving altitude decompression disorders.

PROBLEMS OF SPACE BIOLOGY, VOL. 25: DECOMPRESSION DISORDERS

Introduction

Man's conquest of the deep sea, the upper atmosphere and /7*
outer space entails for his organism the effect of a great number of extraordinary external factors. One of these factors is decompression. In speaking of decompression, some people have in mind only a more or less pronounced reduction of atmospheric pressure, i.e., rarefaction of the atmosphere surrounding man. This, however, is only one type of decompression effect. By "decompression" should be understood any sudden change (drop) from higher to lower in the ordinary pressure of the medium surrounding man, whatever the absolute magnitude of this change may be.

One of the very serious physiological consequences of decompression is the formation in the organism's internal environment of free bubbles of one or another inert gas normally dissolved in the blood and tissues, with which the organism becomes supersaturated as a result of decompression. The symptom complex of the functional disorders connected with the formation of gas bubbles in the organism incident to decompression is customarily called "decompression disorders."

The problem of decompression disorders arose almost 130 years ago when, at the suggestion of the French engineer Triget, laborers working under water in the Forties of the last century began using caissons: watertight bottomless substructures which are sunk to the bottom of a body of water and from which the water is expelled by forcing compressed air into them. This method enabled man to work on the bottom of the sea under conditions of an air atmosphere all right, but one having an increased pressure corresponding to the depth of the submerged caisson. The return to ordinary conditions of a man after working in a caisson, which is accomplished by means of air locks, is inevitably connected with a decompression effect.

Spreading quickly and widely, the caisson method of working /8
under water at once brought out the symptom complex of the specific functional disorders arising in people upon emerging from caissons. The most typical symptom of these disorders was joint and muscular pains localized in the large joints of the extremities and adjacent regions and often enough reaching excruciating proportions. This explains the origin of the folk designations for these symptoms: "courbatures" in French, "bends" in English, "zalomay" ["breaking up"] in Russian. But since decompression in

* Numbers in the margin indicate pagination in the foreign text.

the early days of caisson work was brought about without any precautions -- at one point even needlessly rapidly --, decompression disorders did not remain limited to muscular and joint pains but assumed more serious forms like acute and often fatal disorders of the circulation and respiration, as well as pareses and paralyses of the extremities and entire lower part of the body. The whole symptom complex of disorders that caisson work brought to light received the name "caisson disease."

Soon after the introduction of caissons, the development of diving showed that this symptom complex of disorders is of paramount importance to this type of underwater work, too. The rapid return of divers to the surface led to the same serious, and often enough irremediable and fatal, consequences.

The cause of these terrible manifestations being then unknown, blindly were different compression and decompression methods tested, some people even going so far as to propose versions in which compression was effected slowly while decompression was deliberately brought about rapidly. For his ignorance of the true cause of decompression disorders man paid heavily in human suffering and mass sacrifices. It was only in the Eighties of the last century that the true nature of decompression disorders was brought to light in the classical investigations of Paul Bert but no more than 20 years later the studies by J. Haldane laid the scientific foundations for their prophylaxis. The fundamental results obtained by P. Bert and J. Haldane were embellished, expanded and worked out in detail in a great number of studies by other authors. As a result, light was shed on many aspects of the problem of decompression disorders and, as applied to the conditions of diving and caisson work, relatively satisfactory measures for their prophylaxis (gradual decompression), as well as methods for their treatment (recompression), were worked out by the experimental method.

At the end of the Thirties and the beginning of the Forties of the present century, the problem of decompression disorders acquired new urgency in connection with the rapid development of aviation and the rise in flight ceilings. Upon ascending to altitudes in excess of 8 - 9 km (using oxygen), fliers developed muscular and joint pains and collapsoid conditions. However, in /9 spite of the obvious similarity of altitude decompression disorders to the decompression disorders in caisson and diving practice, and in spite of the existence of straightforward theoretical premises for correct interpretation of these disorders, it was typical that their nature should at first seem obscure. In particular, people came out with hypotheses about their ischemic etiology and other erroneous suggestions. But, as compared with the original problem, the period of guesswork and errors was short. The decompression nature of altitude pains and altitude collapses incident to a normal supply of oxygen to the organism very soon became evident.

This was assisted by numerous experimental investigations stimulated by questions of aviation practice and aimed at studying why and how altitude decompression disorders develop and what to do for their prophylaxis. The problem of prophylaxis, too, as it applies to the requirements of aviation, was solved rather rapidly and, it would seem, quite satisfactorily. It was established that even comparatively short-lived (within the limits of one hour) breathing of pure oxygen before ascending to an altitude (desaturation from nitrogen) rather effectively prevented the development of decompression disorders in high-altitude fliers. Thus it seemed that the problem of the prophylaxis of altitude decompression disorders had already been solved in its entirety.

This idea was dispelled by the development of astronautics and space physiology. There arose the new problem of the prophylaxis of decompression disorders in astronauts when emerging from their spacecraft into empty space or onto the surface of atmosphereless heavenly bodies, which can be done with the aid of spacesuits in which a low total pressure is created. In this case, two new circumstances appeared that had no parallel in flying practice: the need for a man to stay in his spacesuit for a long time, amounting in some cases to many hours, and the need for executing at the same time one or another type of possibly even very strenuous muscular work. Under these two conditions, as the very first investigations showed, the prophylaxis of decompression disorders by preliminary, comparatively short-lived, oxygen breathing, which was usual in aviation, turned out to be ineffective. Nor did increasing the periods of desaturation (oxygen breathing) even up to many hours yield the desired resultsunder these conditions. This being so, it became clear that decompression disorders (muscular and joint pains) can develop a very long time after a transition to a low pressure -- in the third or even fourth hour of a stay in a rarefied atmosphere --, inspite of the continued breathing of pure /10
oxygen. Thus, in connection with astronautics, the whole problem of altitude decompression disorders appeared in an unexpected new light, and the problem of their prophylaxis on spaceflights stood out with particular clarity. At the present time this problem is still in the scientific research stage.

And so the problem of decompression disorders crops up in four broad compartments of special human activity: diving and underwater construction work in caissons, aviation and astronautics. In the historical sequence in which they developed, these types of activity, which are aimed at conquering the deep sea, the upper atmosphere and outer space, in themselves determined the entire course of scientific research on the problem of decompression disorders. The history of the scientific resolution of this problem, which crops up in diving as well as in aviation and astronautics, will be sketched in appropriate sections of this book. Here, however, in the introduction, we must dwell on three more important circumstances that characterize the development of the problem of decompression disorders.

The first is that in working out the given problem, researchers were dominated by practical problems posed by life: preventing decompression disorders and safeguarding the corresponding type of work. In the case of very urgent practical problems, the empirical way of solving them is often faster and more efficient than thorough and in-depth scientific research. Investigation of decompression disorder prophylaxis did indeed follow an empirical course, as a result of which the theoretical development of the problem, and in particular, the working out of its physiological side, lagged very far behind. Thus, if we take the example of diving and construction work in caissons, we will see that the decompression methods used at the present time, based on the general scientific principles established by Haldane, in their details were worked out purely empirically -- by practical approbation of different calculated versions. But the calculations turn out to be very abstract -- in the first place, because there is a lack of concrete data on the saturation of the different tissues of the organism with inert gases, their desaturation from these gases and the conditions under which gas bubbles are formed and, in the second place, because the physiological principles governing the development of decompression disorders has been studied but little. Further, if we retrace the unraveling of the problem of decompression disorder prophylaxis in aviation, as we did above, it will turn out that the altitude decompression disorders that were brought to light ushered in a whole new wave of theoretical studies; but desaturation from nitrogen by breathing pure oxygen was the accepted method of decompression disorder prophylaxis in fliers and had become current practice, while the aforesaid theoretical /11 studies have not yet received a logical conclusion to this day. Moreover, the prophylactic measures adopted in practice, as we have already said, turned out to be not absolutely reliable or above reproach as far as being thoroughly and deeply grounded in science is concerned. Finally, if we turn to the very urgent problem of decompression disorder prophylaxis in astronauts on space flights, which has just cropped up in recent years, we will find a similar state of affairs. Practice really needs to be issued immediate recommendations on the prevention of decompression disorders for the case where astronauts step out into space and is goading researchers to pursue an empirical solution of this problem by testing different methods of transition to a low pressure. In the course of these studies, moreover, it has become clear that without experimental analysis and elucidation of a great number of questions of long standing that are now cropping up again, it is difficult to count on any valid and radical solution of this problem.

The second very important circumstance that to some extent predetermines the empirical character of research on decompression disorder prophylaxis is that from the very beginning and down to the present time, the main and practically only criterion for the development of decompression disorders has been and is muscular and joint pains. All prophylactic measures for the prevention of decompression disorders have been and are being worked out only on the basis of this criterion (sometimes taking into account some

other, milder, yet more reliable, subjective signs of decompression disorders, such as, for example, an itching skin, paresthesia, etc). It is remarkable that man has now been acquainted with decompression pains for more than 130 years yet still does not know where -- in which formations and tissues -- the gas bubbles that cause these pains are localized. This fact is a clear indication of how inaccessible to study are the inner causes and mechanisms of decompression disorders. Often, in speaking about the insufficiency of criteria for the development of decompression disorders, the first idea that comes to mind is that pains are a subjective index, on the basis of which it is generally risky to draw conclusions. This is indisputably a groundless idea and ensues only from the fact that people who assert it are insufficiently acquainted with the matter of decompression disorders. In the first place, by properly selecting our examinees, we can rely on their testimony completely; in the second place, there are ways of checking up on the correctness of subjective testimony in those cases where it is open to doubt (a change in pressure in the altitude chamber that was not expected by the examinee); in the third place, there is always the possibility of a self-experiment and personal verification of the results and, finally, in the fourth and most important place, sensations of pain are accompanied, if not from the very beginning, then soon thereafter, by a number of outward, clearly noticeable, objective expressions. Such expressions are an abnormal position and disturbance of the motor function of the ailing extremity, outwardly noticeable changes in the general condition of the organism and a number of vegetative indices (change in pulse, respiration, etc) that accompany the pains. All these objective signs -- the equivalents of subjective sensations -- are reliable criteria for the development of decompression disorders. It is a reliable basis for drawing conclusions about how to carry out experiments on people and the only basis for chronic experiments with decompression disorders in animals. The fact that the painful decompression syndrome is a reliable criterion for decompression disorders is also borne out in practice by the effectiveness of the prophylactic measures worked out on their basis. So the trouble is not that decompression pains, like all pains, are a subjective phenomenon. The inadequacy of this criterion for decompression disorders is determined by other circumstances. It consists in the fact that the painful decompression syndrome, as this is now becoming indubitable, does not reflect the beginning of decompression phenomena, i.e., the beginning of gas formation in the organism. It already corresponds to a later stage of this process. Consequently, it gives no basis for judging of the appearance in the organism and the quantitative evolution of that internal factor which is the immediate cause of decompression disorders. This raises an extremely serious question: are not all measures of decompression disorder prophylaxis that have been worked out on the basis of this criterion palliative-symptomatic measures, and if so, how can radical-etiological decompression disorder prophylaxis be ensured and established on a firm scientific foundation? The only way to answer

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this question is by in-depth study of the physical bases and physiological principles of development of decompression disorders.

The third circumstance that characterizes the stated problem is the considerable disconnectedness that can be observed in the study of its two fundamental aspects: on the one hand, diving and, on the other, aviation and astronautics. In spite of the fact that, as we have already said, the decompression nature of altitude pains was established very soon after their discovery, this aspect was to a considerable extent studied like a problem apart, bereft, as it were, of any natural and due connection with studies of decompression disorders connected with diving. This state of affairs prevails down to the present time. There is every reason to assert that many specialists in the field of underwater physiology, who are extremely versed in the problem of decompression disorders as applied to diving and are even engaged in their study,/13 are most poorly acquainted with the principles of development of altitude decompression disorders. Similarly, many specialists in aerospace physiology, including those who study altitude decompression disorders, are poorly acquainted with the very rich experience and extensive experimental data concerning decompression disorders in diving practice. This disconnectedness in working out the two principal aspects of decompression disorders is manifested in the fact that scientific research centers and their experimental stations are, as a rule, oriented in either one or another of these two directions and equipped only with either compression or vacuum altitude chambers. This same disconnectedness appears in publications, too, as well as in the separate organization of conferences and symposia, where either the one or the other aspect of decompression disorders is discussed. This state of affairs is to a considerable extent impeding the development of the problem as a whole and its theoretical foundations inasmuch as there can be no doubt that the same physical and physiological principles are at the bottom of all cases of decompression disorders. The differences and distinctive features of decompression disorders arising after a stay under increased pressure and incident to a transition to a rarefied atmosphere should undoubtedly be an object of study for the proper specialists. But it is precisely by confronting these distinctive features that we will probably be able to succeed in establishing the general physiological principles of decompression disorders. The elucidation of these general principles will, in turn, facilitate our understanding of the specific character and distinctiveness of the decompression disorders encountered in different spheres of special human activity.

PART 1

GENERAL ASPECTS OF DECOMPRESSION DISORDERS

CHAPTER 1

PHYSIOLOGICAL EFFECTS OF DECOMPRESSION AND PRACTICAL SIGNIFICANCE OF DECOMPRESSION DISORDERS

In order to gain a better understanding of the practical /14
importance of decompression disorders , it is necessary to analyze in greater detail the decompression effects encountered in practice and the possible physiological consequences of these effects. As we have already said, man runs into decompression, i.e., a change from higher to lower in the ttotal pressure of the surrounding medium, in four special domains of his activity: diving, construction work in caissons, aviation and astronautics.

Incident to diving and construction work in caissons, decompression effects are always preceded by an increase in pressure of the medium surrounding the organism. A man's work under water or in a caisson is always performed under conditions of increased pressure, decompression being an absolutely inevitable effect upon his return to normal conditions after this work.

It should be noted that diving can also be performed in watertight constructions in which a gaseous medium of the usual composition and pressure is maintained. To this class of constructions belong the so-called rigid diving suits, as well as bathyscaphes or bathyspheres -- steel chambers that can withstand the very high pressure effects of the surrounding aqueous medium while maintaining the usual atmospheric pressure within. Using these apparatus, it is possible to plunge to very great depths of the sea. In all these cases, however, man finds himself completely isolated from the aqueous medium surrounding him and can fill the role of no more than a passive observer of what is presented to his field of vision through the windows with which said apparatus are provided. Mechanical manipulators controlled by a man with the aid of mechanisms located inside his heavy diving suit or bathyscaphes are as nothing compared with a man's unrestrained activity at these depths and do not in principle alter his role of passive observer /15
of external phenomena transpiring beyond the window. Thus, all these constructions can serve the purpose of getting to know sea life at very great depths, but they preclude a man's unrestrained activity in the aqueous medium.

To the class of constructions that completely protect man from the effect of the aqueous medium also belong submarines. In these, too, a normal atmospheric pressure is maintained and these, too, preclude a man's unrestrained activity in the surrounding

aqueous medium. But a submarine fleet does not set itself such goals, it faces other problems. If, however, we have in mind a man's emergence from a submarine into the surrounding aqueous medium (and this is possible with the aid of the air locks existing on submarines) and subsequent return (ascent) to the surface, in this case the effect on the organism of the concomitant increased pressure and subsequent decompression is quite inevitable. Such situations are created, in particular, when crew members return to the surface from sunken submarines. Thus, any watertight underwater construction with a normal pressure of the atmosphere filling it precludes a man's unrestrained activity in the surrounding aqueous medium. Here we do not have in mind the so-called underwater houses or underwater laboratories in which an increased pressure corresponding to the depth of their location is maintained and which ensure the free emergence of their inhabitants clad in appropriate underwater accoutrement into the surrounding aqueous medium and their return.

Unrestrained activity under water is possible only when using light diving suits ensuring a man's free movement and action in the aqueous medium. But, independently of the design of such suits, their use is connected with the effect on the organism of the overpressure of the surrounding aqueous medium and of the corresponding overpressure of the inspired air or gaseous mixture. These uneliminable and obligatory working conditions of the diver under water retain their force even when using deep-sea diving equipment provided with a roomy, spherical, copper helmet in which the man's head is housed. When this type of equipment is used, the lower parts of the body of the diver located at a certain depth in a vertical position are squeezed by the water, while the upper part of his body and, in particular, his head are in a gaseous medium having an overpressure equal to the pressure of the surrounding aqueous medium.[1] Consequently, the diver's organism as a whole /16 experiences an effect of increased total pressure corresponding to the depth at which he is located, and incident to any change in this depth, it is subjected to the effect of a corresponding change in the total pressure. Return to the surface and normal conditions is inevitably connected with a decompression effect. The same thing happens when caissons are used. Entering a caisson and emerging therefrom through air locks are also connected with an

[1] Here I do not wish to enter into the details of a certain difference that exists in the absolute magnitudes of the pressure on the lower and upper parts of the body of a vertically standing diver, which is due to the height of the water column at the corresponding levels. Here it is important to note in this connection only that the pressure of the air or gaseous mixture in the space under the helmet must always be somewhat higher than that of the water column at the level of the head and must correspond to the water pressure at the level of the upper part of the breast.

increased pressure and decompression effect. And so, incident to diving, as well as construction work in caissons, an increased pressure effect on man and subsequent decompression effects are inevitable.

And how do matters stand in aviation and astronautics? Here man's activity is transfered from ordinary conditions to conditions of a rarefied atmosphere or complete vacuum. Airtight aircraft and spacecraft cabins can prevent decompression effects on man. But it would be erroneous to believe that decompression effects in aviation and astronautics thereby lose their significance and topicality.

In the first place, for a great number of reasons it proves to be expedient to maintain reduced pressure conditions in airtight aircraft and spacecraft cabins. In aviation, this turns out to be a generally accepted rule: as an aircraft climbs, the pressure in its cabin is reduced, in which case a certain gradient (0.3 - 0.4 atm) is maintained between its value in the cabin and in the surrounding atmosphere. In civil aviation at flight ceilings of 8 - 9 km, the pressure in aircraft cabins is reduced to a level corresponding to 3 - 3.5 km; in military aviation, which reaches the greatest possible altitudes, the reduced pressure limit in aircraft cabins corresponds to an altitude of 8 km (267 mmHg), in which case crew members will of course breathe through oxygen masks. This reduction of the pressure in aircraft cabins is dictated not only by technical circumstances, but also by the need to prevent the catastrophic consequences of extremely strong decompression effects in the case of cabin depressurization. Utilized in the cabins of present day spacecraft are different versions of an artificial gaseous atmosphere. Utilized in Soviet spacecraft at the present time is an ordinary air atmosphere with a normal pressure; in American spacecraft, a pure oxygen gaseous mixture with a pressure of 258 mmHg, which corresponds to an altitude of about 8 km. Promising is the utilization in spacecraft of a two-component gaseous atmosphere with a reduced total pressure and a normalized oxygen partial pressure. Thus, airtight aircraft cabins and, in a number of cases, spacecraft cabins, too, do not eliminate decompression effects or the effect of reduced pressure, /17 but merely keep them within permissible limits.

In the second place, decompression effects in aviation and astronautics are very important because there always remains the danger of an accidental (emergency) or forced (premeditated) depressurization of the aircraft or spacecraft cabin. Sudden cabin depressurization in upper atmosphere layers and, a fortiori, under the vacuum conditions of space, can be accompanied by so-called explosive decompression, which is fraught with especially serious consequences. In view of precisely this possibility of destruction of all means of protection and, consequently, the danger of decompression and action of the surrounding vacuum -- a danger

which constantly accompanies the astronaut on his flight and which can never be completely removed --, some researchers have called the decompression factor the Achilles' heel of space flight.

Finally, in the third place, in the case of astronautics, stepping out of a spacecraft cabin into empty space or onto the surface of an atmosphereless heavenly body in a light suit is inevitably connected with a decompression effect on the astronaut inasmuch as the pressure in his suit is lower than in the spacecraft cabin. In an oxygen atmosphere with a reduced pressure, such as is used at present in American spacecraft cabins, the decompression effects on the astronaut as he changes to his suit are not considerable or dangerous. A pure oxygen atmosphere, however, even on condition that it have a significantly reduced pressure, must not be regarded as a physiologically optimal version of an artificial gaseous medium even for relatively short-lived space flights. But as far as the contemplated superlong-distance flights are concerned, there is every reason to suppose that under these conditions, such an atmosphere will generally be unacceptable. In the case of an air atmosphere with a normal pressure, such as is used in Soviet spacecraft, the decompression effect on a suited astronaut as he steps out into empty space are more considerable. These effects can be reduced by using in spacecraft a two-component gaseous atmosphere with a moderately reduced total pressure and a normalized oxygen pressure. Such an atmosphere, as was pointed out above, is promising for practical utilization. If, however, we turn to the problem of superlong-distance space flights, physiological recommendations dealing with the gaseous medium in spacecraft will probably again be directed toward a normal or close-to-normal atmosphere.

In the third part of this book, these questions dealing with the problem of forming an artificial gaseous atmosphere in spacecraft will be examined in greater detail. Here it was necessary to touch upon them in order to stress that the decompression /18
effects connected with stepping out into empty space are now and will continue to be a very important factor in space flights that include extravehicular activity on the part of the astronauts, and in themselves constitute a topical problem of space physiology and medicine.

The functional disorders arising under the action of decompression effects can belong to three essentially different categories of phenomena.

The first is an increase in volume and pressure of the gases in those body cavities that do not have completely free communication with the surrounding atmosphere. To such cavities belong the stomach and the intestine, where there is always a certain amount of gases, the air-containing nasal accessory sinuses (frontal sinus and maxillary sinuses) and cavities of the middle ear. In the event of decompression effects of a certain intensity, equalization

of the gas pressure in these cavities with the environment does not succeed in taking place, and the pressure in the cavities turns out to be higher than that of the environment. In the unyielding bony cavities, the physiologically active factor is this overpressure acting on the mucosa of the cavities, on the vessels and on the nerve endings and causing sensations of pain and reflex functional shifts as a result of mechanical pressure on the receptors or of ischemia. In the stomach and intestine, decompression leads to a greater or lesser increase in volume of the gases they contain. Here the physiologically active factors are the gas overpressure and the stretching of the walls of the intestine and stomach which, in the case of rapid decompression, may exceed the limits of their elasticity and lead to their rupture. In the event of abrupt decompression effects (explosive decompression lasting fractions of a second, which can take place incident to sudden depressurization of aircraft and spacecraft cabins and the submerged part of caissons or decompression incident to the emergency surfacing of divers), in addition to the above-mentioned gas-containing cavities, the lungs also suffer. In these cases, an overpressure is created in the lungs, too, leading to mechanical damage (including rupture) of the pleura, pulmonary tissue and pulmonary vessels. The intrapulmonary air penetrates into the mediastinum, the pleural cavities, through the ruptured vessels into the blood and with the blood into the arterial vascular system; an aeroembolism of the coronary or cerebral vessels can take place and the whole syndrome of barotrauma of the lungs can lead to fatal consequences.

The second category of phenomena in decompression effects is the formation of free gas bubbles inside the organism owing to the nitrogen or other inert gas dissolved in the blood and tissues. Incident to diving or construction work in caissons, during a man's stay under increased pressure his organism is additionally saturated with the inert gas; as a result, incident to the subsequent decompression -- in the present case, incident to return to normal pressure -- the organism becomes supersaturated with this /19 gas, which changes over from a dissolved to a free state, forming gas bubbles in the organism's internal environment. Incident to the decompression effects that are encountered in flying practice and on space flights, i.e., incident to the action on man of a rarefied atmosphere and reduced pressure, the organism is supersaturated with that inert gas (in the case of oxygen breathing, it is nitrogen) with which the blood and tissues were saturated before decompression, which also leads to formation of gas bubbles in the internal environment. The functional disorders connected with the formation of gas bubbles in the organism will be further discussed, and their analysis constitutes the content of the present study.

The third category of phenomena connected with decompression effects, which is inherent only in the effect on the organism of an abrupt rarefaction of the surrounding atmosphere, is the boiling

of the organism's fluid media and the formation of pockets and hollows filled with water vapors and gases. For the development of these phenomena, the decompression factor itself (in the sense of a drop in pressure, and the magnitude and rate of this drop) is of no very great significance.

The essence of this phenomenon consists in the fact that with a drop in the surrounding pressure down to 47 mmHg (an altitude of 19.2 km), water begins to boil at a temperature of 37°C, i.e, at body temperature; and the boiling point of body fluids is close to that of water. No matter how rapidly or slowly the pressure surrounding the organism is reduced down to the indicated value, as soon as this limit is passed, the body fluids start boiling. This process develops not everywhere, but only in those regions of the organism where the water vapors that are being formed can occupy a certain volume. The conditions most conducive to this accumulate in the subcutaneous cellular tissue. There it is that pockets and hollows are formed (so-called altitude subcutaneous emphysema); this same thing can happen in the intermuscular interstices, in the abdominal cavity. Conditions that are conducive to boiling are also set up in the venous blood of the venae cavae and, during diastole, in the heart cavities inasmuch as the blood pressure here can drop below that of the surrounding blood. In the arterial vessels of the small and, especially, large circulatory systems, provided blood pressure is maintained owing to heart action, there should be no boiling of the blood. Incident to the boiling of body fluids under the indicated conditions, vapor formation and condensation are slightly reversible processes; therefore, with increasing external pressure, the pockets and hollows arising because of vapors are quickly liquidated. It is obvious that this takes place in the blood as its pressure increases upon passing over to the arterial vessels, and this very fact explains the rapid liquidation of altitude tissue and subcutaneous emphysema with increasing pressure of the medium surrounding man. /20
Experiment has shown that in the event of short-lived exposures to the low pressures that make body fluids boil, this phenomenon, at first glance so extreme from the physiological point of view, in itself does not threaten the organism's life. But we must take into consideration the fact that vapor formation in the organism's liquid media and pliable tissues is immediately complicated by the discharge into the hollows that are being formed of gases that have been in a dissolved state in the blood and tissues. At those degrees of rarefaction that we are talking about (not to mention a vacuum environment), the organism is always in a state of supersaturation with gases; supersaturation under these conditions already sets in owing to the oxygen and carbon dioxide dissolved in the organism's media even in the absence of any inert gas, and in the presence of such a gas, it turns out to be even greater. Thus, the process as a whole now acquires a mixed character, i.e., boiling and vapor formation are combined with gas formation in solutions supersaturated with gases.

The three categories of phenomena that we have just examined in connection with decompression effects on the organism have their counterpart in certain functional disturbances or disorders. In principle, they could all be called decompression disorders. By the expression "decompression disorders," however, it is customary to designate only those functional disturbances that are connected with the formation of gas bubbles in the organism's internal media because of its supersaturation with inert gases as a result of decompression. As will be shown below, this has some justification on terminological grounds. Thus, the decompression disorders to which the present study is devoted embrace all the functional disturbances connected with gas bubble formation in the organism as a result of its supersaturation with inert gases incident to decompression, whether we are talking about decompression from increased to normal pressure or from normal to reduced pressure.

We must say a few words about the practical significance of the problem that we have just discussed as compared with the other categories of functional disorders that we mentioned above in connection with decompression effects. As far as the increase in pressure or volume of the gases in the different body cavities incident to decompression is concerned, these phenomena have practically no significance for normally executed diving or caisson work. Even in the event of very rapid decompression, the pressure in the nasal accessory sinuses and cavities of the middle ear of healthy people becomes easily equalized with the surrounding pressure.

Pressure and volume changes in the gases of the stomach and intestine have even less significance for divers and caisson workers. With an increase in surrounding pressure, the gases in the gastro- /21
intestinal tract contract, and with decompression, they return to their initial volume. Meteorism as a result of decompression can arise only in those cases where considerable amounts of new gas were formed and retained in the gastrointestinal tract of the worker during his shift under increased pressure. Generally speaking, in diving and caisson work mechanical consequences of decompression involving the air-containing body cavities have a practical significance only in emergency situations (most often emergency surfacings by divers), when, as a rule, barotrauma of the lungs is the first thing to appear. Nor, apart from emergency situations, is the decompression effect on the air-containing body cavities of any greater practical significance in aviation and astronautics. A somewhat greater role may here be played by altitude meteorism, which, by the way, usually becomes pronounced only when the recommended diet is broken. But in general, as in the case of underwater work, in aviation and astronautics decompression effects on the air-containing body cavities are practically significant only in the event of the excessively rapid and abrupt (explosive) decompression that takes place incident to sudden aircraft or spacecraft cabin depressurization. Once again, in the explosive decompression syndrome, barotrauma of the

lungs (of the intestine, too, perhaps) occupies a prominent position, but, apart from that, under conditions of an acutely rarefied atmosphere and, a fortiori, a vacuum, this syndrome precludes the boiling of body fluids as well as formation of gas bubbles in its internal environment. Besides, we must not forget the decisive significance, under these conditions, of the hypoxemic factor. Thus, for diving and caisson work as well as for aviation and astronautics, functional disorders connected with decompression effects on air-containing body cavities are of practical significance only in emergency situations; the same can be said of the boiling of body fluids under the effect of low pressures.

If now we turn to the functional disorders connected with the formation of gas bubbles in the organism incident to decompression, i.e., strictly to the problem of decompression disorders, we can see that its practical significance is incomparably greater. Indeed, under diving and caisson conditions, decompression disorders pose a constant threat to man upon his return to normal conditions from increased pressure. The current prophylactic measure of gradual decompression gives no absolute guarantee of preventing these disorders. This has been confirmed in very practice: decompression disorders are still encountered in diving and, especially, caisson /22 work, even when all of the current decompression rules have been observed to the letter. But this is still not the main point.

Getting ahead of myself, I must say that the outwardly completely satisfactory cases of gradual decompression of divers and caisson workers that have taken place without any painful symptoms visible functional disorders, are undoubtedly very often, and possibly in most cases, accompanied by the formation of gas bubbles in the organism's internal environment. This will be borne out by the experimental data presented below. Consequently, the internal etiological factor that leads to decompression disorders, in the face of existing methods for their prophylaxis, are very often not entirely removed, but merely kept below certain thresholds. In other words, prophylactic decompression methods as currently applied in diving and caisson practice do not eliminate the possibility of latent forms of decompression disorders. The organism's apparent well-being may turn out to be essentially impermanent, and the threat to its well-being may not have been removed. This is how matters stand in ordinary daily diving and caisson practice. A new branch of underwater activity that is undergoing extensive development at the present time is connected with the problem of the national economic utilization of marine riches and the conquest of the pre-Continental shelf. This is connected with the creation of habitable underwater houses and man's penetration to ever greater depths that hitherto seemed inaccessible. Facts have shown that as applied to these new conditions, the principles and means of decompression disorder prophylaxis used in ordinary diving practice and caisson work are not justified and prove to be inadequate. Consequently, another exceptionally important aspect of decompression disorders makes its appearance incident to diving.

In aviation, for flights that take a normal course, decompression disorders in their common acceptation do not in practice constitute a critical problem. But should we penetrate more deeply into the heart of the matter, we would find that the problem of decompression disorder prophylaxis under flying conditions have not been radically solved, either. The facts show that a rarefaction of the atmosphere corresponding to an altitude of 8 km (such as takes place in the cabins of altitude aircraft), is dangerous from the point of view of decompression disorders. Recent experimental data show that the comparatively short-lived desaturation of the flier's organism with nitrogen while breathing oxygen that is prescribed by the instructions prevents decompression disorders from developing only provided the flier performs no muscular work, and most altitude flying itself lasts a comparatively short time. We have only to /23 apply a physical load under conditions of rarefaction corresponding to an altitude of 8000 m and prolong the stay under these conditions for decompression disorders not to be long in making themselves felt. Hence, it follows that decompression disorder prophylaxis in pilots of altitude aviation is not absolutely reliable and that this problem cannot be regarded as being either of little significance or completely solved.

The exceptional importance of decompression disorders for astronauts is clear from what has already been said above. Space walks by astronauts will undoubtedly be a necessary and frequent operation in future space flights. This being so, space physiology and medicine are being squarely confronted with the problem of decompression disorder prophylaxis, which is only now being worked out experimentally.

It should be noted that everything we have just said about the importance of the problem of decompression disorders in diving, construction work in caissons, flying and astronautics refers to planned, if one may say so, human activity that takes a normal course under these conditions. This determines the paramount topicality of the problem of decompression disorders and compels us to distinguish these disorders from all of the other functional disturbances that we have examined above, which are connected for the most part with accidental decompression. Nor must we forget that in the event of accidental decompression in all of the enumerated spheres of activity, the decompression disorders that constitute the subject of our discussion come into manifest effect.

In concluding this chapter, I must say a word about terminology. To designate the functional disorders arising in people incident to decompression as a result of the formation of free gas bubbles in their organism, many different terms have been proposed. The oldest are caisson disease, diver's disease, compressed air disease. These cannot be acknowledged as being successful because, in the first place, the functional disorders referred to, as we have already said, can arise not only in caisson workers and divers, but also in fliers and astronauts and can be connected not only with

the effect of compressed air, but also with the effect of atmospheric rarefaction. In the second place, these disorders as a whole, if we bear in mind all of their manifestations, must not be identified with disease. This question, which is a matter of principle that goes beyond the confines of terminology, will be examined at the end of the book on the basis of the factual material presented there. Instead of the above terms, M. I. Yakobson [1950] proposed to call the functional disorders connected with excessively rapid transition of the organism from a higher to a lower pressure, "aeropathy." This term, intended by the author to unite all cases of free gas in the organism's internal environment and all the functional disturbances connected therewith, did not come into currency. An obvious shortcoming is that it /24 embraces the most diverse pathologic conditions connected not only with the formation of gas bubbles inside the organism, but also with their entry into the organism from the outside (for example incident to traumas, surgical interventions, etc). Thus, the term "aeropathy," on the one hand encourages identification of decompression disorders with disease and, on the other, does not single out their etiological characteristics. In the foreign literature of recent years that is devoted to decompression disorders in fliers, to designate these disorders the term "aeroembolism" has been widely employed. It accurately reflects the essential pathogenetic component of decompression disorders not only incident to the action of a rarefied atmosphere, but also incident to transition from increased to normal pressure. But inasmuch as the whole symptom complex and all of the manifestations of these disorders cannot be reduced merely to aeroembolism, the given term is not acceptable, either.

Recently, the term "decompression sickness" or "decompression illness" has become current. These terms decidedly apply to the manifestation and consequences of decompression effects in all of their diversity of symptoms and seriousness, beginning with a slight postdecompression itching of the skin and ending with the paralysis and sometimes sudden death of people after return from under increased pressure. It goes without saying that any disease may be accompanied by symptoms of different intensities and take a more or less serious course, and it is quite legitimate for investigators to want to embrace under one term consequences of decompression disorders that differ in their degree of manifestation. The terms "decompression sickness" and "decompression illness" are not acceptable to me because a great number of functional shifts incident to decompression effects, as will be shown below, are in their essence not morbid pathologic symptoms, but defense reactions of the healthy organism in response to an unusual effect. In a word, my disagreement with the given terms is based on the principle that certain states of the organism arising incident to its interaction with unusual factors differ from disease or sickness.

On the basis of all of the foregoing, the most practical of all the existing terms are the terms "decompression disorders" or "decompression disturbances." These terms are not free from

shortcomings, either; excessively rapid decompression can lead to functional shifts not only in virtue of the formation of gas bubbles in the organism's internal environment, but also as a result of the expansion of gases contained in body cavities and the boiling of body fluids at high degrees of atmospheric rarefaction. However, inasmuch as each of these cases has its own designation (barotrauma of the lungs, altitude meteorism, altitude emphysema), it has become widely accepted to connect the /25
term "decompression disorders (disturbances)" only with functional shifts caused by the formation of gas bubbles in the organism's internal environment. Here, however, it becomes imperative to make still another terminological distinction. The facts show that postdecompression gas formation in the organism, so long as it does not reach a certain intensity, does not lead to the development of the typical symptoms or outwardly noticeable functional shifts. It goes without saying that it would be incorrect to call such cases "decompression disorders" or "decompression disturbances;" they must somehow be classified otherwise. For this I will use the term "decompression phenomena," and for the conditions resulting from return from under increased pressure, the term "caisson phenomena." These terms will designate all those cases where gas bubbles are formed in the organism as a result of its supersaturation with inert gases, regardless of whether or not this process causes any noticeable changes. But those cases where the development of decompression phenomena in the organism is accompanied by symptoms of one kind or another, will be designated by the standard terms "decompression disorders" or "decompression disturbances."

CHAPTER 2

HISTORICAL DEVELOPMENT OF THE PROBLEM OF DECOMPRESSION DISORDERS

As has already been pointed out, the problem of decompression disorders acquired independent significance in the middle of the last century in connection with the wide dissemination of construction work under water. The descents under water that had been made up to this time in bells, as well as in primitive diving suits, were not numerous and did not show at all clearly the typical consequences of rapid decompression after a stay under increased pressure. So the effect of these conditions aroused no general interest at that time inasmuch as no mass diving took place and such as did, had no direct practical significance. After Triget in 1839 - 1841 successfully used in the Loire valley the caisson method he proposed for sinking coal shafts, this method was rapidly disseminated and became widely employed in different countries for the construction of bridges and underwater tunnels. This being so, decompression disturbances immedia- /26
tely emerged as the typical, frequent and very dangerous consequence

of those unusual effects to which caisson workers were subjected. A great number of terrible postdecompression muscular and joint pains, pareses of the extremities, irremediable paralyses of the lower part of the body, and sudden respiratory and circulatory disorders, very often terminating in death, became the constant companions of any more or less extensive construction work by the caisson method. But inasmuch as the symptoms of decompression disturbances developed in far from all workers and since the caisson method was irreplaceable in its field, the use of this method kept spreading; the army of caisson workers grew, and so did the number of casualties.

The first scientific publication devoted to decompression disturbances appeared in the medical literature in the year 1854. Its authors -- the French physicians Pol and Watelle -- reported on their observations of 64 caisson workers and described the development in these workers, after emerging from caissons, of pathologic manifestations and symptoms that they themselves designated by the term "caisson disease." These observations showed that the probability that caisson disease will develop and the seriousness of its morbid symptoms increase with increasing pressure, working time in the caisson and decompression rate; the latter circumstance gave the authors grounds to recommend, as early as this initial study, a slow reduction of pressure incident to the emergence of workers from a caisson, i.e., to point out that measure which, as became clear subsequently, is the only radical one for decompression disorder prophylaxis. This recommendation in the study of Pol and Watelle, however, had a purely empirical character inasmuch as the authors gave a completely erroneous interpretation of the cause of decompression disturbances. They evolved a theory of mechanical compression, according to which the functional disturbances in caisson workers occur in consequence of a shift of the blood from the periphery to the depths of the body when working under pressure and its inverse redistribution upon emergence from the caisson, i.e., when the surrounding pressure decreases. So dominated were the minds of researchers and physicians by these incorrect ideas that the early study of Robert Boyl [1670], who had observed the appearance of gas bubbles in the ocular fluids of an adder incident to rarefaction of the atmosphere and had pointed to gas formation in body "juices" as a possible cause of the death of animals under these conditions, remained forgotten; consigned to the same oblivion was a similar report by Van Musschenbroek [1739]. Even those researchers who themselves had noted the possibility of gas formation in the organism incident to rapid reduction of the surrounding pressure [Hoppe-Seyler, 1857; Bucquoy, 1861; Mericourt, 1869], were unable to break away from the theory of mechanical compression. An exhaustive critique of this theory was given by V. V. Pashutin /27
[1881], but even after this, in explaining some phenomena connected with a change in general pressure, individual researchers still turned to it; what is more, even in our time the idea is sometimes

revived among physicians (of course, not those specifically occupied with the effect on the organism of a change in pressure), that the peripheral vessels of the body contract incident to compression and dilate incident to decompression. V. V. Pashutin correctly explained the tenacity of these ideas by the circumstance that man very often encounters a local pressure effect on the surface of his body from different mechanical causes, when the peripheral cutaneous vessels are inevitably compressed; hence the natural attempt to transfer this obvious causal effect to different cases of changes in the general pressure of the medium surrounding man. Apparently, this same circumstance explains the fact that the explanation of caisson disorders proposed by Pol and Watelle, in spite of the fact that they were not tested experimentally and in many respects did not even agree with observations, nevertheless became generally accepted. Medical thought was in bondage to these incorrect ideas for many years and was freed from this bondage only by the capital investigations of P. Bert [1878], the brilliant initiator of scientific experimental treatment of the problem of decompression disorders. In speaking of these investigations, we must remember that their conceptual bases were to a considerable extent foreshadowed by the studies of the French physician Jourdanet. The latter treated a number of cardinal physiological aspects of the effect of a rarefied atmosphere on the organism quite correctly, and considered it necessary to test his conclusions experimentally. Being a well-off man, Jourdanet subsidized the building of high and low pressure chambers for P. Bert, who did not have sufficient means.

In spite of ourselves, we must marvel at what P. Bert did for science in his short span of life.[2] He analyzed in detail the effect on the organism of increased and reduced air pressure, revealed the true causes of the functional changes that this entails, was the first to investigate the effect of oxygen want, discovered and studied its toxic effect under pressure, obtained cardinal data about the effect on the organism of limit carbon dioxide concentrations and, finally, carried out a careful investigation of the mechanism of decompression disturbances. The results of these latter investigations still constitute down to this day the basis of scientific concepts about the nature of decompression disorders. These disorders, as was established by P. Bert, arise as the /28 result of the fact that gases from the inspired air, and especially nitrogen, which were dissolved in the organism's blood and tissues during the effect of increased pressure, do not, incident to rapid decompression, succeed in escaping from the organism by diffusion but form in the organism's internal environment free gas bubbles

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[2] In 1886 P. Bert was sent on a government mission to Indochina and died of dysentery that same year in Hanoi at the age of 53.

which, depending on their localization, form morbid symptoms of one kind or another. In addition to laying down this general principle based on extremely comprehensive experimental material, P. Bert solved a great number of very important questions of a more particular kind; nor have these concrete factual data lost their value down to this day, and researchers studying individual aspects of decompression disorders are constantly obliged to turn to them. These data include: a concrete description of the outward picture of decompression disturbances in human beings and different animals; results of studying the gaseous composition of the blood incident to the action of increased pressure, showing that the dissolving of gases in the blood incident to compression basically obeys Dalton's law; results of determining the composition of the gas bubbles that are formed in the organism after decompression; comparative data about the resistance of different animals to decompression disturbances, etc. Finally, the exceptional value of P. Bert's work resides in the fact that it was crowned by practical, scientifically grounded recommendations on the prevention and therapy of decompression disorders. The expediency of slow decompression was experimentally demonstrated, the use of oxygen for theraputic purposes during the development of decompression disturbances was legitimized, and at the same time the principal and only radical method of eliminating them was described: therapeutic recompression.

In this way physicians and engineers overseeing and organizing caisson operations acquired correct ideas about the nature of decompression disorders and general recommendations for combatting them. This was not long in telling on practice: the number of cases of decompression disturbances, especially their serious forms, was appreciably reduced, and the outcome of these cases, thanks to Bert's radical therapeutic measures, became more favorable. But the practical result turned out to be far from as significant as might have been supposed. The reason for this is that left unresolved in P. Bert's investigations was the important question of principle which was subsequently elucidated by J. Haldane Sr.: at what degree of supersaturation of the organism with nitrogen do gas bubbles begin to form? To P. Bert were available data indicating that after a man's stay under a pressure of 1 - 1.2 psig and less, decompression disorders never developed; but he did not make use of these facts to treat the above-mentioned question. As a result, the practical recommendation of slow decompression, which/29 was perfectly correct in principle, did not culminate in a concrete expression with a theoretical and experimental foundation. This task was accomplished by J. Haldane only 30 years later. In the meantime, researchers and diving physicians, guided now by P. Bert's work, continued to accumulate factual material on the problem of decompression disturbances. To this period belong studies abounding in practical observations contributed by Russian diving physicians: P. S. Kachanovskiy [1875, 1881], Ye. M. Khrabrostin [1888], F. I. Shidlovskiy [1894], N. A. Yesipov [1902]; the theoretical investigations by N. Zuntz [1897] aimed at characterizing the process of

saturation of the organism with nitrogen during a stay under increased pressure; the studies by A. Layet [1876] devoted to the significance of temperature conditions in the development of decompression disorders; the capital generalizing work by R. Heller, V. Mager, and H. Schrötter [1900]; the very important study by H. Vernon [1907], showing that the solubility of nitrogen in fats is considerably greater than its solubility in water; and the beginning of the investigation by L. Hill and M. Greenwood [1906, a, b; 1907, 1908] devoted to the principles of saturation of the organism with nitrogen and its desaturation from this gas.

At the end of the last and beginning of the present century the significant and rapid advances of diving were studied in different countries. In this period new types of diving suits and respiratory apparatus were built, the whole individual diving technique was much perfected, and diving itself now became a profession and acquired the role of a special service with practical significance to the nation as a whole. The increasing of diving depths emerged as the main concern of diving specialists, and in this connection the question of decompression disorder prophylaxis after a man's stay under pressures much greater than those characteristic of most caisson work, stood out with new sharpness. Attempts to descend from the shallow depths that had been mastered to deeper depths were being accompanied by numerous cases of decompression disorders in divers, showing the inefficiency of existing prophylactic measures and the necessity of improving them cardinally. This problem was successfully solved by J. Haldane Sr. with a group of assistants: the physician Boycott and diving specialists, an officer in the Royal Navy by the name of Damant and Warrant Officer Katto [Haldane, 1906; Boycott and Damant, 1908; Boycott, Damant and Haldane, 1908].

Haldane drew attention to the above-mentioned fact that decompression disorders never set in, if the pressure under which a man finds himself (even for a very long time) does not exceed 1.25 psig. Experiments on animals and investigations of people con- /30 firmed this fact and led to the important conclusion of principle that the organism's blood and tissues possess the ability to retain nitrogen in the state of a supersaturated solution up to a certain supersaturation limit; only when this limit is trangressed does the formation of gas bubbles in the organism begin and decompression disorders develop. This conclusion constitutes the principal achievement of Haldane and his coworkers, and it ensured the practical results of their work and to a considerable extent determined the entire subsequent development of the problem of decompression disorders. Haldane and his coworkers came to the conclusion that the maximum permissible saturation of the organism with nitrogen incident to decompression is determined not by the absolute magnitude, but by the rapidity of the drop in pressure. The thesis they advanced that if decompression from 2 to 1 atm is safe, then decompression from 4 to 2 atm and from 6 to 3 atm should also be safe, has been confirmed by experiments on animals and investigations of people.

As a measure of maximum permissible supersaturation, Haldane proposed a coefficient showing the ratio of the nitrogen pressure in the organism's tissues after decompression to the partial pressure of the nitrogen in alveolar air.[3] If this coefficient, when a man returns from increased to normal pressure, is exceeded in none of the organism's tissues, decompression, according to these views, should be safe.

Another great achievement of Haldane and his coworkers was the theoretical constructs they developed for the purpose of determining the nature and rate of saturation of the organism's different tissues with nitrogen under increased pressure conditions and of their desaturation from this gas incident to decompression. The authors came to the conclusion that, in general outline, the nature of the saturation and desaturation processes in the organism as a whole and in its individual tissues is the same and can be expressed by a logarithmic curve showing that 50% saturation is reached in approximately 1/5 of the time necessary for complete 100% saturation. Subsequently, experimental data from different authors showed that these theoretical considerations were close to the truth. As far as the rate of saturation and desaturation is concerned, it was clear to Haldane that it must differ sharply for different tissues of the organism, depends on a great number of physiological conditions and is determined in the first place by the blood supply in the tissue. Being unable to study this question experimentally (so complicated are the methods involved in this problem that it has not yet been solved to this day), Haldane proposed to get his bearings from two extreme cases of saturation (desaturation) of the tissues: the fastest and the slowest, and a number of intermediate cases. For extreme cases, the concrete /31 rate of saturation could be assumed on the basis of certain facts. Such facts for the case of fastest saturation were data of Hill and Greenwood on the rate of saturation of human urine with nitrogen under increased pressure conditions. On the basis of these data, Haldane and his coauthors assumed that the semisaturation time of the tissues that become saturated most rapidly comes to 5 min. The starting point for slowest saturation was the fact that the danger of decompression disturbances is directly dependent on the length of the organism's stay under pressure within the limits of a six-hour period; but if exposure exceeds this period, the danger of decompression disorders does not increase. This gave grounds to assume that the semisaturation time of the tissues that become saturated most slowly comes to 75 min. The choice of intermediate cases was arbitrary. Haldane and his coworkers confined themselves

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[3] Subsequently, A. P. Brestkin [1952] introduced an important correction into the notion of this coefficient: it is theoretically more correct and practically more convenient to determine the coefficient as the ratio of the nitrogen pressure in the organism to the total external pressure after decompression.

to three such intermediate cases with tissue semisaturation times of 10, 20, and 40 min respectively. On the basis of all of these data, Haldane and his coauthors calculated gradual decompression methods for depths down to 62 m.

Haldane's methods sharply reduced the frequency of cases of decompression disturbances in diving and caisson practice. Serious decompression disorders became a rarity, and fatal cases were relegated to the category of extraordinary events connected with infringement of established decompression rules. This practical effect best of all illustrates how the principles adopted by Haldane to calculate his decompression tables were fundamentally correct, and how close to the truth were his conclusions about the conditions under which decompression disturbances develop. These fundamental principles in their general outline are still valid today.

At the same time, very soon after the introduction of Haldane's methods into practice, it became clear that they were not ideal. In spite of applying these methods, decompression disturbances of an intermediate and, especially, of a low degree remained fairly common occurrences. Subsequently, as diving depths became greater, the inadequacy of the calculation method proposed by Haldane for decompression conditions became all the clearer. None of this, however, is meant to diminish Haldane's merit as the founder of scientifically grounded decompression disorder prophylaxis.

It should be noted that the studies of Haldane and his coworkers, in addition to their tremendous positive significance, also had some negative consequences. These consequences boiled down to two circumstances. The first is that the utilization of abstract theoretical tissues with different saturation and desaturation rates that Haldane proposed, which was completely justified in calculating decompression methods detracted the attention of researchers /32 from concrete saturation and desaturation processes in real tissues of the organism. It turned out that the proposed calculation method to a considerable extent freed researchers from the necessity of experimentally studying how the organism's different organs and tissues become saturated and desaturated; which of them present the greatest threat of gas formation, and under what circumstances do they do so; where is the first place in the living organism that gas bubbles are formed after decompression; and how do they develop their pathogenic effect. Striving to meet practical demands as rapidly as possible, Haldane did not stop to analyze in detail the physiological principles underlying the development of decompression phenomena and focused his attention only on the chief general conditions under which pronounced decompression disorders develop. He succeeded completely in his practical aim, but by this very success he willy-nilly detracted the attention of researchers from analysis of the intimate mechanism of decompression disturbances. Ensuring the conquest of ever greater depths and

working out decompression disorder prophylaxis as applied to the new diving conditions, researchers followed more or less the same path as Haldane, and for the most part did not deal with the properly physiological side of the problem; but with the passage of time, the lack of knowledge in this area became ever more apparent, and now there can be no doubt that without in-depth study of the physiological principles governing the development of decompression disorders, it will never be possible to perfect their prophylaxis.

The second circumstance with a certain negative effect ensuing from the investigations by Haldane and his coworkers on the subsequent development of the problem of decompression disorders was that the authors, in checking their calculations, passed judgment on the maximum permissible supersaturation of the organism on the basis of the outward symptoms of decompression disturbances. Hence arose and subsequently gained strength the idea that the appearance of gas bubbles in the organism is equivalent to the appearance of morbid symptoms and, vice versa, the absence of symptoms means the absence of gas bubbles. In other words, in testing and working out their decompression methods, researchers after Haldane came to assume that the absence of decompression symptoms means that in no tissue of the organism did supersaturation reach the critical level and that, consequently, the calculations carried out on precisely this assumption were fully justified. Below it will be shown that these ideas are profoundly erroneous.

After Haldane's investigations, the problem of decompression disorders developed very intensively and was now stimulated almost exclusively by the growing requirements of diving practice. For construction work in caissons, which is carried out as a rule at /33 depths not exceeding several dozen meters, the decompression methods that were worked out turned out to be generally satisfactory. But the development of diving posed ever newer problems connected with further increases in diving depths and length of stay on the bottom and, simultaneously, with the need under these conditions to reduce the decompression period as much as possible, i.e., to ensure maximum diving efficiency.

In accordance with these requirements, the overwhelming majority of all more or less large-scale investigations of the problem of decompression disorders carried out after Haldane has a decidedly practical cast and is aimed at perfecting means and methods of decompression. And at the same time, in a number of these investigations, as well as in individual special studies, theoretical questions were also subjected to study.

A great number of authors studied the principles of dissolution of different gases and, in particular, nitrogen, in different fluids in vitro, as well as in different fluid media and tissues of the organism [Quincke, 1910; Hawkins, Shilling & Hansen, 1935; Behnke & Jarbrough, 1939; Scholander & Edwards, 1942; Whitely et al, 1944; Brestkin, 1952]. The attention of researchers was constantly

attracted to the question of the principles of saturation of the organism as a whole and of its different media and tissues with inert gases and their desaturation from these gases [Campbell & Hill, 1933; Behnke et al, 1935, 1941 a, b;Kreis, Pavlovskiy & Prikladovitskiy, 1936; Kandror and Shik, 1939; Kandror, 1940; Ardashnikova, 1948; Lawrence, Jones, Berg, Franklin & Loy, 1948; Tobias,et al, 1949; Brestkin, 1952; Lundin, 1953]. A good deal of attention was devoted to studying those circumstances under which gas bubbles begin to form in the media of the organism that are supersaturated with an inert gas [Piccard, 1941; Dean, 1944; Harvey, et al, 1944 a, b, c; Harvey 1950; Whitaker et al, 1945; Catchpol & Gersch, 1946, 1951; Yakobson, 1950; Savichev & Bukharin, 1950; Malkin, 1951; Brestkin, 1952; Balakhovskiy & Fomenko, 1956; Bukharin, 1958]. Data began to pile up about the morphological changes in the central nervous system incident to serious and lethal forms of decompression disorders [Zografidi, 1907; Blick, 1909; Bohrnstein, 1910; Stettner, 1911; Nordman, 1928; Egoleton, et al, 1945; Rozcakedyi & Soon, 1956].

In practical observations and special studies, the effect was elucidated of different external conditions and of the state of the organism (in particular, the external temperature and muscular work) on the development of decompression disturbances [Tseytlin, 1935; Fraser & Rose, 1942; Whitaker, Blinks, Berg, Twitty & Harris, 1954; Griffin, Robinson, Berding, Darling & Turrell, 1946; Smedal, Brow & Hoffman, 1946; Anthony, Clanke et al, 1951; Aver'yanov, 1960; 1962; Brestkin, Gramenitskiy et al, 1958].

At the same time that different decompression methods for /34
depths exceeding those to which Haldane had confined himself were being feverishly devised therapeutic decompression methods were being empirically worked out. These investigations resulted in much factual material of great value from the theoretical and practical points of view. But throughout the first two decades after Haldane's investigations, the problem of decompression disturbances was in the main treated in terms of detailing the individual partial questions that went to make it up without leading to any general results of significance as a matter of principle.

With the end of the Twenties and the beginning of the Thirties of the present century, this problem went through a new stage of development marked by great and rapid advances in the conquest of hitherto inaccessible depths, as well as by a number of theoretical achievements. This stage was connected with the discovery of the toxic effect of the inert gases, in particular nitrogen, when using helium for deep-sea diving or oxygen for decompression. In this stage, in addition to the studies of foreign investigators, in the first place Behnke and his collaborators, domestic diving physiology underwent extensive development and soon wrested the leading place in the world. The making of this specialized physiology is closely linked with the name of Academician L.A.Orbeli and the scientific activity of Professor M. P. Brestkin.

At the end of the Twenties, under the guidance of L. A. Orbeli, the scientists Ye. M. Kreps, S. I. Prikladovitskiy, B. D. Kravchinskiy and S. P. Shistovskiy undertook studies aimed at safeguarding deep-sea diving. These studies were conducted in close contact with a group of members of a diving expedition with a peculiar name (Epron) headed by Rear Admiral S. I. Krylov with the constant active participation of Chief Physician Epron K. A. Pavlovskiy, Naval Engineer A. Z. Kaplanovskiy and a number of diving specialists.

These studies led to a rapid accumulation of scientific material and a substantial practical achievement. Ye. M. Kreps et al [1936 a, b] studied the distinctive features of saturation of the organism with an inert gas as a function of breathing and investigated the physiological significance of helium; K. A. Prikladovntskyy [1940], and then A. V. Voyno-Yasenetskiy [1947, 1950] undertook an analysis of the toxic effect of compressed oxygen on the organism. In experiments on animals and on themselves, B. D. Kravchinskiy and S. P. Shistovskiy [1936] determined safe oxygen breathing times for different pressures, studied the desaturation of the organism from nitrogen under these conditions and experimentally legitimized the use of oxygen incident to decompression. Within a short time, in conjunction with the use of air for breathing, these newly- /35
developed methods were being successfully applied in diving to depths of 100 m and more.

Studies connected with the problems of deep-sea diving gained especially in scope at the end of the Thirties and beginning of the Forties, when M. P. Brestkin created in the Military Medical Academy a special pressure-laboratory with a unique technology, which made it possible to study the effect of both the low and the high pressures of gaseous media that were the maximum permissible ones for that time.

By this time the practice of diving had led to the discovery of the toxic effect of nitrogen under pressure and had shown that a depth of 100 m is the limit for using compressed air to breathe. In this connection, new diving methods with helium-oxygen mixtures were beginning to be worked out, and new decompression methods with gradual switching over of the diver's breathing at a depth of 60 m from the helium-oxygen mixture to air, and in the final stages of decompression, from air to pure oxygen. This study, which was conducted under laboratory as well as marine conditions, under the general guidance of L. A. Orbeli and with the participation as leading specialists of M. P. Brestkin and Ye. M. Kreps, in a very short time led to record results: as early as 1941, for the first time in the history of diving, safe descents down to depths of 200 m were made in light equipment. In addition to its practical results, the study led to serious theoretical results concerning the effect of high nitrogen and helium pressures on the organism. In experiments by L. A. Orbeli and M. P. Brestkin, B. D. Kravchinskiy, K. A. Pavlovskiy and S. B. Shistovskiy [1944] on animals, the toxic effect of nitrogen under increased pressure was studied and the use of helium under these conditions was experimentally legitimized.

Thus, in no more than a decade, diving and the special branch of physiology responsible for its successes had not only advanced in the Soviet Union to the foremost ranks; it had left them far behind.

It should be stressed that such rapid advances in this field were to a considerable extent foreshadowed by the studies of the founders and most prominent representatives of domestic physiology and pathology: I. M. Sechenov, I. P. Pavlov, V. V. Pashutin, P. M. Al'vitskiy, Ye. A. Kartashevskiy, N. V. Veselkin. The originality of Russian physiological thought, and the wealth of generalizations that distinguish it, armed domestic physiology with those general principles which must be made the foundation of any study of human activity under changed conditions. These general principles of equilibration of the organism with the external environment, principles which pointed to fantastic adaptive resources, and which play the role of nervous system in the organism's interaction with changed conditions, found a direct application in/36 diving physiology.

In addition, the studies of these scientists, in spite of the fact that none of them was dealing specifically with diving problems, placed into researchers' hands extensive factual material, rich in generalizations and having immediate reference to these problems. In this connection, let us mention I. M. Sechenov's studies devoted to gas transport by the blood and to the role of carbon dioxide in regulating breathing, and his theory of the constancy of composition of the alveolar air with formulas permitting accurate calculation of this composition for any change in the surrounding pressure; the data of N. I. Pirobov [1825] and V. V. Pashutin [1881] on the organism's ability to endure intravenous administration of large quantities of air; the very pithy generalizations of V. V. Pashutin on the effect on man of a decrease or increase in the total pressure of the surrounding medium and on the mechanism of embolic phenomena; the capital investigations by P. M. Al'bitskiy [1911], showing the biological significance of carbon dioxide; his studies [Al'bitskiy, 1883, 1884, 1904], and those of his students Ye. A. Kartashevskiy [1906] and N. V. Veselkin [1913], devoted to the effect of a reduction in the partial pressure of oxygen as well as of small and medium concentrations of carbon dioxide. As practice has shown, these factors are in themselves of exceptional significance in diving, and play a great role in decompression disturbances.

Thus, specialized physiology in our country began to develop on a deep conceptual and rich factual foundation. Its distinctive feature was the coupling of the practical purposes of research with profound theoretical interpretation of the results of this research.

After the first dazzling successes in the matter of conquering greater depths in the pressure-laboratory, a wide front was opened

under the guidance of M. P. Brestkin to study the effect on the organism of the different factors entering into a changed gaseous medium. By this time another powerful factor had appeared to stimulate study of decompression disturbances: the latter constituted a serious obstacle to the development of combat aviation by setting limits to increases in the altitudes of flight ceilings. On the example of decompression disorders and other effects of a changed gaseous medium, experiment was beginning to show ever more clearly that the conquest of the deep sea and of the upper atmosphere were very often accompanied by the effect on the organism of the very same factors. Simultaneous study of these factors as applied to the one field or the other, as investigations conducted in the pressure-laboratory had shown, is of great scientific and practical interest, facilitating more in-depth and thorough elucidation of the mechanism underlying their physiological effect. M. P. Brestkin and his coworkers undertook a many-sided and detailed analysis of the effect on the organism of a reduction in the partial pressure /37 of oxygen and explained the exceptionally great role of carbon dioxide in hypoxemic states [Brestkin, 1952, Panin, 1944, 1953, 1958, 1963; Zvorykin, 1951; Sulimo-Samuyllo, 1952; Vasilenko, 1953, 1955; Zagryadskiy, 1954, 1958; Vinokurov, 1958; Vinogradov, Gramenitskiy and Oblapenko, 1955; Gramenitskiy, Sulimo-Samuyllo and Tripolov 1955; Zan Dok Men, 1958, etc].

A special series of studies was devoted to in-depth study of the toxic effect of compressed oxygen [Zhironkin, 1940, 1955; Panin, 1957, 1960; Gramenitskiy & Sorokin, 1958; Sorokin, 1960]. In the course of these studies, in addition to analysis of the mechanism underlying the toxic effect of oxygen under pressure, the distinctive features of its effect on the organism under conditions of physical stress were elucidated. The desaturation of the organism from nitrogen while breathing oxygen under pressure was investigated [Brestkin A. & Zhironkin, 1959] and, rounding out similar data of other authors, the diffusion of nitrogen through man's integuments under these conditions was studied and accurately determined in a quantitative manner. The continued working out of decompression methods for deep-sea diving showed the necessity of in-depth theoretical and experimental study of caisson phenomena, to which indeed a whole series of special studies was devoted. These studies began with the fundamental investigations by A. P. Brestkin [1950, 1952 a, b], in which the principles governing the formation of gas bubbles in different supersaturated solutions in vitro were studied and theoretically explicated, the different abilities of the organism's different media and tissues to retain inert gases in a state of supersaturated solutions were established to be a fact of fundamental importance and the immediate conditions under which the formation of gas bubbles in the organism can be liquidated were elucidated. These investigations yielded concrete data that were the starting point for calculating decompression conditions and stimulated the study of the physiological principles governing the development of caisson phenomena in the organism and,

in particular, constituted a powerful incentive to present research. Beginning to be studied were respiratory and circulatory reactions in serious forms of caisson phenomena in animals as a function of the intensity of gas bubble formation in their blood [Vavilov and Gramenktskiy 1958], as well as the functional shifts arising during the development of decompression symptoms under conditions of rarefaction and the role of some additional conditions in the development of decompression disorders [Aleksandrov 1949; A. Brestkin et al 1958].

These investigations pointed to the exceptional importance of the organism's physiological reactions during the development of caisson phenomena and to the need for further study of this question. The aggregate of these studies conducted in the pressure-laboratory was a powerful spur to the development of the different divisions of special physiology. These advances were due not only to the /38 accumulation of voluminous new experimental material and the solution of a great number of concrete questions of great scientific and practical significance, but also to those theoretical generalizations which were made on the basis of all this research. Studied in the pressure-laboratory, in addition to the factors of a changed gaseous medium, were various other unusual external effects (overloads, rocking, the effect of shockwaves and unusual temperature conditions), and the effect on the organism of muscular work of different, including limit, intensities under usual and changed external conditions was investigated in detail.

The aggregate of these factors led to the conclusion that in all these cases, the organism's activity is subject to the same general principles. Thus, research showed that the individual branches of specialized physiology arising in connection with the need to ensure different types of human activity under different conditions can essentially be united into one broad branch of physiological science which can be called "the physiology of the organism under extraordinary conditions." The main theoretical content of this branch of physiology, which determines its specificity and independent scientific interest, resides, as M. P. Brestkin has pointed out, in the general principles of mobilization of the organism's exceptionally extensive, many-sided and powerful physiological reserves, which it acquired in the course of evolution and the process of ontogenesis. Under the ordinary and, a fortiori optimal, conditions of man's daily activity, these latent reserves of the organism do not come to light, but when extraordinary external factors are encountered, they reveal themselves in all their amplitude, showing the organism's superlative ability to actively resist their harmful effect and adapt itself to new, often very difficult, conditions of activity. In spite of the motley nature of these external factors, the organism, in interacting with them, switches on and utilizes its emergency resources in accordance with the single general principle that in the first stage of this interaction, everything is aimed at defending the organism from the harmful effect of the changed conditions and at supporting and

strengthening those physiological functions and those types of activity that are of decisive biological significance at that moment at the same time that other functions and other types of activity that play a secondary and unimportant role in the given situation are sometimes very drastically inhibited. This principle is the key to correct interpretation of the different physiological changes that take place in the organism under the effect of extraordinary factors. It shows that in a great number of such cases, the physiological shifts that at first glance seem purely pathological disorders are actually indispensible components of the organism's general defense reaction and determine its resistance to harmful effects. /39

This principle explains why the reflex activity of the central nervous system under the action of powerful extraordinary factors, contrary to wide-spread ideas, is switched on not in stages -- from the higher to the lower centers of the brain -- but in response to whatever function of the organism is involved; why, under the extraordinary circumstances that the organism has encountered, we have the unique picture of all the nerve centers that control a given physiological system being operative in that system, while not only the conditioned, but also the unconditioned reflexes of other systems turn out to be suppressed; thus, the functions of the brain prove to be stratified not vertically in stages, but horizontally in individual systems that control this or that activity. Study of the physiological effect of the most diverse external factors has shown that when these factors are repeated not too strongly, the organims's physiological reserves increase, and its reactions aimed at mobilizing these resources become ever more thorough; this embodies the basis of the organism's adaptation and conditioning to activity under changed conditions.

The general principles of interaction between the organism and extraordinary factors that we have necessarily been expounding in a most compressed form are not only of theoretical, but also of practical significance inasmuch as they arm researchers with the correct approach to the different questions of specialized physiology that arise in practice and enable their most rapid, scientifically well-founded solution. It should be stressed that the tendency to unite the individual trends of specialized physiology into one independent physiological discipline with its own theoretical bases usefully distinguishes the development of this branch of science in our country from its status in other countries, where the study of the corresponding specialized physiological questions is of a narrower, more practically applied, nature.

In the last few decades, the problems of deep-sea diving and decompression disorders, simultaneously with their study in the pressure-laboratory of the Military Medical Academy, have been treated by representatives of a great number of other institutes.

In particular, in 1941, N. V.Lazarev, on the basis of experiments on the biological effect of different inert gases under pressure, published a study aimed at systematizing the information about this problem. The different physiological and hygienic questions connected with the problems of diving and underwater swimming were being systematically and fruitfully studied in the Department of Specialized Physiology of the Military Medical Academy. Here, /40 under the leadership of Ye. E. German, materialized a number of studies and dissertations devoted to the effect of increased air pressure on different functions of the organism [Alekseev, 1945, 1957; Fokin, 1954; Myasnikov, 1954, etc] and dealing with the problem of decompression disorders [Zarakovskiy, 1957; Aver'yanov, 1960, 1961, 1962, 1964; Aver'yanov, Gramenitskiy, Savich, 1961; Aver'yanov and Zarakovskiy, 1966; Pestov, 1960; Yunkin, 1965]. In 1955, under the editorship of Ye. E. German appeared "Uchebnik spetsial'noy fiziologii" [Manual of specialized physiology], in which, on the basis of data in the literature and studies by the authors themselves, the fundamental physiological and hygienic problems of diving and, in particular, the problem of decompression disorders, were systematically set forth. This book was useful in virtue of not only its direct educational purpose, but also its further scientific development of the given branch of physiology, inasmuch as in systematically expounding the subject, the gaps in our understanding of the different problems involved became especially apparent. The insufficiency of existing information about the mechanism of decompression disturbances stood out particularly clearly.

Specifically devoted to the problem of decompression disturbances are two monographs: M. I. Yakobson's "Kessonnaya bolezn'" [Caisson disease] [1950] and A. N. Bukharin's "K voprosy o profilaktike i lechenii kessonnoy bolezni" [On the prophylaxis and therapy of caisson disease] [1958]. The positive significance of M. I. Yakobson's book resides in the fact that it is the first book to be openly published at home in which the extensive material in the literature on the problem of decompression disorders was collected and systematized. But the author, as will be shown in detail below, developed completely erroneous ideas about the conditions under which decompression disturbances develop and their mechanism, for which reason his book not only did not advance the study of these cardinal problems, but actually sent researchers down the wrong path in solving them. The same, unfortunately, must be said of A. N. Bukharin's monograph, in which the author treats the experimental material he obtained from a preconceived and erroneous point of view, and enunciates incorrect principles on how to work out prophylactic methods of decompression. These principles will be discussed below in the light of the experimental checking they received in the present study. In 1961, G. L. Zal'tsman's monograph "Fiziologicheskiye osnovy probyvaniya cheloveka v usloviyakh povyshennogo davleniya gazovoy sredy" [Physiological bases of man's stay under conditions of increased pressure

of a gaseous medium], in which the author discusses the problem as a whole in general outline, paying particular attention to the question of which of the artificial gaseous mixtures used in diving are the best from the standpoint of composition. The idea underlying the author's research on optimal mixtures was, in the first place, for the gases to be used in the combinations and at /41 the concentrations that had the least toxic (narcotic) effect under high pressure conditions and, in the second place, for the danger of decompression disorders to be lessened and for decompression time to be reduced by enriching these mixtures with oxygen. The author's research in this direction resulted in theoretically interesting material on the effect on the organism of nitrogen and helium under pressure and yielded essentially practical results. It showed that by using air-oxygen mixtures instead of air in diving to intermediate depths, the length of safe decompression methods can be reduced several times, and that by using air-helium mixtures instead of helium-oxygen mixtures, the undesirable effects caused by helium at high pressures can be lessened. In that book the author does not take up the study of the physiological principles governing the development of decompression disturbances. But the elucidation of this question that he did undertake on the basis of data in the literature showed in all clarity that this question had been studied quite insufficiently.

The foreign literature of the 1950 - 1960's, too, was enriched with many studies dealing with the problem of decompression disturbances. A considerable number of them were devoted to the study of individual aspects of the etiology and pathogenesis of decompression disorders, as well as their prophylaxis and therapy.

Some of these studies constitute synopses; to this category belongs the reliable book of Haymaker [1957], in which the author, on the basis of very extensive material in the literature and his own research, discusses in detail the pathogenesis of decompression disorders and the serious types of morphologic changes that accompany them in the central nervous system; synoptic, too, is the article by Plummer [1964], which is not distinguished by an abundance of the material the author uses but which contains well-known data about the bases of diving physiology. Many studies constitute a description of different cases of severe decompression disturbances, or such as took an unusual course [Haymaker, Davison, 1950; Haymaker, Johnston, Downey, 1956; Obland, 1959; Steward, 1961; Laba & Gwozdziewicz, 1964; Slark, 1964; Goodman, 1964]. One of these studies stands out: that of Rivera [1964], which contains a detailed analysis of 935 cases of decompression disturbances in divers and data about the effectiveness of the therapeutic recompression methods adopted by the USA Navy.

The abundance of studies of diving physiology that have been carried out in the last few decades reflects the intensive and many-sided development of this field of knowledge,

In connection with the complication of diving conditions, the practice of diving poses a great number of new questions dealing with the effects of different depth and decompression factors. Inasmuch as these questions allow of no delay, they are to a considerable /42 extent solved empirically, and their theoretical side, because of the complexity of working them out, is inevitably lagging behind in its development.

In the problem of decompression disorders, this theoretical side of the matter, dealing as it does with the mechanism of their development, turns out to be especially complex; therefore, in spite of the abundance of accumulated factual material, our knowledge in this field remains far from complete.

CHAPTER 3

FUNDAMENTAL PHYSIOLOGICAL ASPECTS OF DECOMPRESSION DISORDERS

Raised in the present chapter are fundamental questions concerning the conditions for and mechanism of development of decompression disorders. Before considering them, the following excuse is in order. In the first place, these questions will here be discussed from the standpoint of how matters stood before the beginning of the experimental research presented below. This serves the purpose of directing the reader to those aspects of the problem that will be elucidated in the present book, and of placing on a scientific footing the logic behind this experimental research.

The questions raised will be the subject of further discussion; it will be necessary to return to them repeatedly in connection with the facts adduced and the latest data in the literature, so that they can finally be given an up-to-date interpretation. In the second place, I did not deem it necessary to discuss the physicomathematical side of the studies and research dealing with the genesis and evolution of gas bubbles in supersaturated solutions and media. Theoretical analysis of this side of the matter should be left to the competent specialists. Therefore I will not deal with mathematical computations and formulas but will confine myself to bringing up for discussion only those fundamental deductions and conclusions to be drawn from the studies in question that are of significance as principles explaining the etiology and developmental mechanism of decompression disorders. But, thirdly and lastly, I do deem it necessary to dwell separately in the present chapter on those conceptual and experimental premises that determine the direction of research involving physiological analysis of decompression disorders.

The first important question about decompression disorders is as follows: Under what conditions does the process of

postdecompression formation of gas bubbles arise in supersaturated solutions in general and in the media of the organism in particular? /43

According to ideas originating with P. Bert and much consolidated after Haldane's studies, the only important condition for the genesis of postdecompression gas formation in the organism's media is for these media to be supersaturated with nitrogen or some other inert gas. But it should be pointed out at once that this stand, apart from the general data from which P. Bert and Haldane derived it, is supported by solid theoretical foundations as well as by experimental confirmation [A. Brestkin, 1952, 1958]. But the most important thing is that these ideas do not in any apparent manner contradict the vast experience that has been acquired with diving. This is why these ideas are shared by a preponderant number of diving physicians and diving specialists to this day.

But at the end of the Forties of this century ideas in the sphere of altitude physiology began to take shape about the conditions under which gas bubbles are formed in the organism's fluids and media incident to decompression that differed in principle from the foregoing. These new ideas were developed by Harvey and his coauthors in analytical studies of the developmental mechanism of altitude decompression disorders, which at that time were one of the most pressing problems in aviation. So acute had this problem become by 1942 that the USA government and National Science Board that year set up a special subcommittee to study altitude decompression disorders, and it was as members of this subcommittee that Harvey and his colleagues conducted their scientific and experimental research. It must be said that this research was distinguished by rare extensiveness. As early as 1944, in the first three issues of the Journal of Cellular and Comparative Physiology there appeared six publications by Harvey and his coauthors devoted to the conditions and mechanism of gas-bubble formation in the organism incident to altitude decompression [Harvey, Barnes, et al, 1944; Harvey, Whiteley, et al, 1944; Harvey, Mc Elroy, et al, 1944; Mc Elroy, Whiteley, Cooper, et al, 1944].

In the course of 1945 - 1947 four more communications by the same group of authors [Harvey, Barnes, et al, 1945; Harvey, Whiteley, Cooper, et al, 1946; Harvey, Cooper, et al, 1946; Harvey, Mc Elroy and Whitely, 1947] came out, and subsequently, in 1951, all of this material, thanks largely to the efforts of Fulton, was correlated in the collection: Decompression Sickness. This material contains interesting data and conclusions about altitude decompression disorders. Also, it was in these studies that the new general ideas about the genesis of gas bubbles incident to decompression that we have been talking about were formulated. The gist of these ideas is that the fact of

supersaturation of a fluid by a gas dissolved in it is of itself not sufficient for gas-bubble formation in the supersaturated /44
solution. According to these ideas, the obligatory condition for gas formation in the organism's fluids or media that are supersaturated with a gas is the presence there of microscopic "gas nuclei," into which gas molecules penetrate, changing these "nuclei" into larger gas cells, which subsequently become the centers of gas formation. Sometimes these views are incorrectly interpreted to mean formation in fluids and tissues of vacuums under certain conditions (of a mechanical order). It is not, of course, a question of vacuums in the literal acceptation of this word, but of microscopic cavities filled with water vapors and gas molecules that are formed in fluid media as a result of mechanical moments (cavitation phenomena). This phenomenon was studied and described in 1894 by the Irish physicist Reynolds. He reproduced it in experiments with glass tubes that had narrowings; water saturated with air gases under ordinary atmospheric pressure was passed through the tubes. As the water flowed along the tubes, cavities and bubbles formed in those places situated immediately beyond the narrowed section. This, as Reynolds showed, was the result of turbulent eddy motions in the fluid connected with the reduction of pressure that the fluid undergoes upon passing from the narrowed to the wide section of the tube (Bernoulli's law). Reynolds showed that at a high enough flow rate of the fluid and a great enough narrowing of the tube, a very low pressure can be attained upon coming out of it, resulting in pronounced turbulence and cavitation, and the naked eye can see intensive formation of cavities and bubbles that maintain themselves in the fluid. Reynolds called this phenomenon "boiling of water in an open dish at room temperature." The Reynolds phenomenon was one of Harvey's main starting points in building his conception. Harvey reproduced this bubble formation under the indicated conditions and filmed it with the aid of high-speed motion picture photography. In addition, there were some other premises for Harvey and his coworkers to build their conception on. Thus, in 1912 Hill in his monograph "Caisson Sickness and the Physiology of Work in Compressed Air" pointed to the existence of "weak points" in a fluid supersaturated with a gas as being the necessary condition for bubbles to form in it. In their studies, the physical chemist and mathematician Kenrick and his coauthors [1924] and Piccard [1941] also talked about gas "nuclei." But it was only in the above-mentioned systematic studies by Harvey and his coauthors that this conception underwent extensive development as applied to altitude physiology.

It should be noted that in building this conception initially on the analogy of the Reynolds phenomenon, the authors emphasized the genesis and existence of gas nuclei in the blood as a result of its possible turbulent motions in individual sections of the cardiovascular system (for example, near the atrial ventricular valves, /45
where the arterial vessels branch out, etc). But in their

subsequent deliberations the authors shifted the center of attention to deformation of microcavities and gas nuclei in the tissues because of contractions of the musculature, which, in their opinion, can cause considerable local reductions of pressure, i.e., create the prerequisites for such microcavities to be formed. Incidentally, this stand, too, is sometimes simplified and misinterpreted to mean literally that in the tissues foci with a negative pressure to tens of atmospheres are formed, forgetting that the pressure of a medium cannot literally be less than zero. So here we are talking about certain relationships among mechanical forces and certain changes in the internal stress in structural elements of tissues that arise in tissues incident to muscular contractions. This can be approximately pictured as, for example, a relative negative pressure in the pleural cavity (without the presence there, as is normally the case, of a gaseous or vaporous medium) or as the kind of change in structural stress in some tissue or material that arises in it when it is stretched and tested for its resistance to rupture. Fulton [1948], wishing to illustrate the nature of this kind of phenomenon, comes up with the following example. He writes: "In gases, the pressure can never drop below zero, but in fluids and dense substances the negative pressure or stress (italics my own. P. G.) can be very considerable. Thus, in Berthelot's experiments, clean, degassed, glass tubes were filled with water at a high temperature; then the temperature was reduced, whereupon a negative pressure of about 150 atm was formed before a cavity was formed in the fluid column."[4] The conditions that are adduced in the given example (a fluid in a closed unyielding space) are no doubt quite different from those that accumulate in tissues incident to different combinations of mechanical forces.

But without entering into these details let us return to the fundamental idea of Harvey and his coauthors that the supersaturation of fluids and tissues does not in itself predetermine the genesis of postdecompression gas bubbles and that their formation for these degrees of supersaturation that are actually encountered in practice is possible only on the basis of preexisting gas nuclei in the organism's media. At the present time, this idea has many adherents and is shared by many authors [Genin, 1951, 1948; Albano, 1965; Nikolayev, 1969, 1970; etc.]. At the time /46 of its formation, however, the initial studies of Harvey and his coauthors had not received any extensive publicity among Soviet physiologists. The idea under consideration found its way into our domestic literature not in a pure form, but in a new and distinctive interpretation. In 1950 M. I. Yakobson's monograph Kessonnaya bolezn' [Caisson disease], in which the author, contrary

[4] Fulton, D., Decompression sickness.] Origin of bubbles in tissues. Cf. The Russian translation in the book Voprosy aviatsionnoy meditsiny [Problems of aviation medicine], page 113.

to the views established since Haldane's time about the maximum permissible supersaturation (coefficient of maximum permissible supersaturation) of tissues as the main and determinitative condition for post-decompression formation of gases in the organism, advanced a new conception that he called the "endogenous-exogenous" theory of the etiology of decompression disturbances. Alluding superficially to Harvey, the author made the hypothesis that the ability of the tissues and blood to retain nitrogen in the state of a super-saturated solution is extremely great and that the supersaturation of the organism with nitrogen, which takes place under actual practical conditions, cannot in itself cause the formation of gas bubbles. In the author's opinion, in order for bubbles to be formed incident to decompression one more condition is necessary: the penetration from without of "gas seeds" into the blood super-saturated with nitrogen. Yakobson assumes that their appearance in the blood supersaturated with nitrogen leads in principle to the same effect as salt crystals thrown into a solution super-saturated with salt: just as in this case intensive precipitation of sediment takes place in the saline solution, just so does violent gas formation take place in the supersaturated blood when "gas seeds" get into it. Yakobson believes that "gas seeds" can get into the blood incident to decompression from, firstly, the alveolar space and, secondly, the intestinal cavity. Guided by an ancient study by Ewald and Kobert [1883], the author believes that under certain conditions the pulmonary tissue is penetrable to air; and he ascribes this same property to the intestinal epithelium. But, in his opinion, the condition leading to the penetration of "gas seeds" into the blood through these natural membranes is an even comparatively small increase in intrapulmonary and intraintestinal pressure, which supposedly almost always accompanies decompression.

Thus, according to Yakobson's views, decompression disorders develop as a result of the simultaneous action of two factors: the one endogenous (the supersaturation of the organism with an inert gas) and the other exogenous (the ingress of "gas seeds" into the blood vessels). It ensues from all of the author's arguments that the latter factor in the final analysis even turns out to be decisive inasmuch as it is necessary for "supersaturation of the organism with a gas to turn into an aeroembolism." Yakobson did not support his conception with any special factual data. He confined himself only to citations from the literature, theoretical arguments and consideration of individual cases of decompression disorders after a stay under increased pressure that, in his /47 opinion, speak in favor of the notions he developed. It is easy to show that Yakobson's conception is in fact not original. If, as has already been said, the author of the idea of the high resistance of solutions supersaturated with nitrogen in the tissues in the absence of "gas nuclei" in them is Harvey, then the conception of the penetration of "gas seeds" into the blood vessels incident to decompression belongs to L. Hill [1912]. Experimenting

on guinea pigs, the latter also found that incident to a high content of gases in the animals' intestine, decompression after a stay under increased pressure is accompanied by earlier and more serious functional disorders. The author recognized the true decompression nature of these disorders and assumed that they cause small gas bubbles to penetrate into the blood from the intestine.

Thus, Yakobson's conception was an amalgamation of all these ideas. But in all fairness to the author it must be said that his monograph was the first in the domestic literature and to this day remains the only solid work devoted to "caisson disease." It contains in a detailed and systematic form extensive material of a statistical nature about the symptoms of decompression disorders incident to emergence from under increased pressure, about internal and constitutional factors that promote their development, about the clinical course of decompression disturbances that differ in seriousness and about therapeutic recompression and other methods of therapy for casualties. In this connection, Yakobson's book is very useful and has lost no significance for the present day. From the very moment of its publication it attracted the attention of the competent specialists to the author's above-mentioned conception of the "endogenous-exogenous" origin of decompression disorders.

Immediately upon its publication Yakobson's book was reviewed by L. A. Orbeli and M. P. Brestkin, under whose leadership at this time in the pressure-laboratory of the Military Medical Academy it was with particular intensity that prophylactic decompression methods for returning from great depths were being worked out experimentally and theoretical investigations of gas-bubble formation processes in solutions supersaturated with gases were being conducted (A. P. Brestkin). The methods were worked out on the basis of the general principles introduced by Haldane. In calculating and experimentally checking these methods, Haldane's principles were subjected to many-sided analysis and received important rectifications, but turned out to be basically correct. Yakobson's conception went counter to these principles. If we were to proceed from it in working out the prophylaxis of decompression disorders, we would have to focus our attention not on the degree of supersaturation of the organism's different tissues, but on the possibility of "gas seeds" getting into the blood and /48 we would have to see to it first and foremost that no relative increase in pressure of the gases in the lungs and intestine took place incident to decompression. The untenability of such ideas was obvious.

The effects of meteorism as well as the possibility of increased intrapulmonary pressure were completely eliminated from investigations of divers incident to decompression, yet incident to needlessly harsh (short in terms of general time) decompression

methods, decompression disorders nevertheless arose. Moreover, at this same time self-experiments were being conducted in the pressure-laboratory by myself and a group of student doctors with ascents to altitudes of 11 - 12 km without preliminary desaturation while breathing oxygen under overpressure in the lungs, without there being any external compensation. In these experiments we set up, as it were deliberately, what were according to Yakobson's theory the conditions for rapid and inevitable development of pronounced decompression disorders. But the participants in this study (incident to a 30-minute exposure to the altitude) developed such disorders far from always and not at all more frequently than in experiments without intrapulmonary overpressure. Further, at this same time A. G. Zhironkin with another group of student doctors was conducting self-experiments in altitude meteorism involving deliberate use of a diet that caused intensive gas formation. The "altitudes" were quite sufficient for the appearance of decompression disorders, but no clear-cut altitude meteorism could be recorded to induce them. All of this and, first and foremost, the experimental practice of decompression disturbance prophylaxis might have been expected to suffice to refute Yakobson's conception. But L. A. Orbeli and M. P. Brestkin reasoned differently -- they deemed it necessary to check and disprove Yakobson's conception experimentally, and in this they were undoubtedly right. Yakobson's book had gone out to a great army of researchers, diving physicians and diving specialists; it could confuse their ideas, and what was needed was no verbal criticism of the conception put forward, but irreproachable factual argumentation showing the untenability of the ideas developed by Yakobson. The experimental testing of Yakobson's conception was entrusted to A. P. Brestkin. On his investigations and on the results of his physiological experiments, in which I took part and which were the starting point for subsequent physiological analysis of decompression disorders, I will dwell below.

Right now we must turn to the following important question of principle concerning the conditions and developmental mechanism of decompression disorders. What to adopt as the measure of maximum permissible supersaturation with what to determine that /49 degree of supersaturation at which the gas bubbles are formed that cause functional disorders?

As was mentioned above, what was adopted as such a measure since Haldane's time was the coefficient determining the ratio of the pressure of the inert gas in the organism to the total pressure after decompression. But some researchers [Behnke, 1937; Savichev and Bukharin, 1958] expressed the need to reconsider this determination of the magnitude of safe or maximum permissible supersaturation that Haldane had introduced. The reason for reconsideration was that the Haldane coefficient, which was safe for low

pressures, turned out to be dangerous incident to descents to greater depths. In empirically working out decompression methods for great depths, people began using the so-called differentiated coefficient, decreasing its value with increasing depth. Thus, safe upon transition to the first halt at a depth on the order of 200 m proved to be the coefficient 1.4 instead of the coefficient 1.8, which was used in the last stages of decompression. Proceeding from these facts, Behnke advanced the hypothesis that safe and critical supersaturation is determined not by the ratio of the pressure of the inert gas in the organism to the surrounding pressure, but to the difference of these values. In conformity with this, Behnke proposed decompression methods whose essence consisted in first effecting rapid decompression until the just-mentioned safe-critical difference was reached, and then bringing about slow continuous decompression so as not to exceed this difference. To all appearances, Behnke's methods did not find such a wide practical application; in any case, in 1955 the author himself admitted that they were groundless. I. I. Savichev and A. N. Bukharin [1958][5] advanced a similar thesis, according to which the maximum permissible supersaturation of the organism with an indifferent gas is determined not by the ratio, but by the difference between the pressure of this gas in the tissues and the manometric overpressure. This value, which the authors called the "limit," on their view undergoes considerable individual variations, but for each individual is a relatively stable and very constant value.

The point at issue is of paramount importance: it is a question of the principal condition determining the genesis of decompression disturbances and the principles on which their prophylaxis must be based. No wonder a heated polemic raged around this question. The whole precedent practice of calculating /50 decompression methods using coefficients differentiated for depth, not to mention logical arguments, spoke against the idea of a limit. The adherents of this idea, however, were able to defend their position because in calculating decompression methods, no matter how this was done, only abstract tissues were dealt with; but the actual course of saturation and desaturation of the organism's real tissues remains unknown. Hence the possibility of interpreting the results of checking decompression methods in any number of ways, depending on one's point of view. Thus, A. N. Bukharin, appraising the data he obtained about the depth of the first halt, insisted that tissues that become rapidly saturated possess the ability to retain nitrogen in solution incident to especially high degrees of supersaturation, by as much as a factor

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5 Indicated is the year of publication of the monograph by A. N. Bukharin, in which the author, being completely solidary with I. I. Savichev, develops the latter's views and endeavors to substantiate these common views of theirs.

of 4. Under this condition, the depth of the first halt could be interpreted as being calculated not according to the coefficient, but according to the limit. At the same time, inasmuch as the actual saturation and desaturation rates of the different anatomical substrates were and still are unelucidated, neither the author nor those refuting him had any data about just which concrete tissues were being talked about. Add to this the complete lack of information about the general physiological principles underlying the development of decompression disturbances, and it becomes clear why the controversy inevitably entered the sphere of abstraction. Thus, nothing than this prolonged polemic could more clearly show how great were the gaps in our knowledge of the true course of decompression phenomena in the organism and the mechanism of development of decompression disorders. During this polemic, G. A. Zal'tsman and his coauthors [1961] carried out research on the maximum permissible supersaturation of the organism with inert gases. This research showed that the difference between the pressure of the inert gas in the organism and the surrounding pressure that corresponds to maximum permissible supersaturation increases sharply with an increase in absolute pressure and decreases with a decrease in it. These facts were direct evidence of the untenability of the views of I. I. Savichev and A. N. Bukharin, according to which the limit for different pressures was constant. If the data about maximum permissible supersaturation adduced by G.A. Zal'tsman are expressed by the coefficient, figures show that the value of the latter decreases with higher pressures and increases with lower pressures. To be sure, these changes in the coefficient that are connected with the absolute pressure are incomparably less considerable than the corresponding changes in the difference. Without discussing whether the limit or the coefficient was right, G. A. Zal'tsman believed that in view of the complexity of the relationships between safe pressure of an inert gas in the organism and mechanical pressure, "it is expedient to use the expression 'value of safe or critical supersaturation of the organism,' leaving undecided the nature of the quantitative dependence." [6] /51
With this position it is difficult to agree because, in the first place, the very concept of value without the measure it determines loses its meaning, and, in the second place, elucidation of the nature of the dependence between the pressure of the gas supersaturating the solution and the total pressure is necessary for theoretical comprehension of postdecompression gas formation.

In the course of the polemic about coefficient and limit, it became clear to the overwhelming majority of researchers that the ideas about a limit were untenable and that it was more correct to direct one's attention to the coefficient. But this it was impossible to prove incontrovertibly without undertaking special

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6 G. A. Zal'tsman. Fiziologicheskiye osnovy prebyvaniya cheloveka v usloviyakh povyshennogo davleniya gazovoy sredy [Physiological bases of man's stay under increased pressure of a gaseous medium], L. Medgiz, 1961, p. 61.

experiments for this purpose. Such experiments were the more necessary as not only had the idea of limit found adherents, but individual diving physicians were even endeavoring to apply it in practice. The results of such experiments will be presented below; getting ahead of our story, it can be said that the experimental data, as was to be expected, concerned the correctness of the ideas about the coefficient as the real measure of the organism's maximum permissible supersaturation with an inert gas and showed the complete untenability of the ideas about a limit.

Elucidation of the principal conditions determining the formation of gas bubbles in supersaturated solutions, as well as in the media of the organism, is extremely important for analysis of decompression disorders but does not by any means disclose the mechanism of their development. This side of the problem, as has been stressed more than once, has been the least studied. In order to talk about the developmental mechanism of decompression disturbances it is necessary, to begin with, to ascertain in which media of the organism gas bubbles are firstly and mostly formed incident to decompression. The data in the literature bear witness to the fact that there is insufficient clarity about this, one might say leading question. No study of the organism's maximum permissible supersaturation with an inert gas throws any light at all on this question inasmuch as the organism's supersaturation is established on the basis of the development of external symptoms of one kind or another; about which are the media of the organism in which gas formation in such cases takes place in the first instance and where the gas bubbles responsible for the development of these symptoms are localized, the researcher can only construct different hypotheses and express guesses of one sort or another. That is how matters have always stood since the very beginning and still do stand in experimental working out of different calculated versions of decompression methods. Thus, the main line of research on the problem of decompression disorders bypasses the question of the mechanism of their development.

The information encountered in the literature about which /52 are the media of the organism in which gas formation begins in the event that the organism's maximum permissible supersaturation is exceeded and where the gas bubbles that cause decompression symptoms of one kind or another are localized, is contradictory. No researcher denies the possibility of postdecompression gas formation within the vascular system, in particular in the venous blood, and many authors, beginning with P. Bert, ascribe to it a very great significance in the development of decompression disturbances. In addition to this, most authors also allow the possibility of extravascular gas formation; in particular, Haldane, in discussing the immediate causes of spinal postdecompression affections, cites as the typical location of gas bubbles the white matter of the spinal cord. This schema is used in many subsequent studies

of a synoptic nature, is transfered without critical analysis from one manual to another and strengthens among researchers the opinion that the typical consequence of excessively rapid decompression is the formation of gas bubbles in tissues rich in lipoids. Some researchers insist that gas formation in the tissues plays a determinative role in the development of decompression disorders. Nims [1951], in particular, develops such a point of view in his theoretical analysis of the developmental mechanism of postdecompression joint and muscular pains. His basic idea is well-founded and extremely simple: in the event that a gas bubble situated in the tissues begins to grow, it exerts pressure upon them and causes their deformation, which leads to stimulation of sensory receptors and causes painful sensations. These effects ought to be the greater, the less yielding the tissues and, of course, the richer they are in nerve endings, which is substantiated by the experiments of Inman and Saunders [1944], who injected Ringer's solution into different tissues of examinees. The experimental data showed that when the solution is injected into the tendons, ligaments, tissues of the joint capsules and, especially, under the periosteum, painful sensations arise at considerably lower values of the overpressure than when it is injected into the muscles and other, less dense tissues. All these effects, which are well-known to practicing physicians and experimenters even without any special experiments and are based on quite intelligible mechanical relations, Nims subjected to careful mathematical analysis, expressing in appropriate formulas that deformation pressure at which the gas bubbles growing in the tissues can, according to the calculation, lead to the development of painful sensations. It is very probable that Nims' conception is close to the truth, but it contains no direct evidence to the effect that the gas bubbles that cause the symtoms actually arise in the tissues.

A very considerable step forward in the sense of amplifying /53 and making more precise our knowledge of decompression disorders was the systematic research conducted in this field by A. P. Brestkin [1952]. On the results of this research I will permit myself to dwell in somewhat greater detail because, in the first place, it was the starting point for further physiological analysis of decompression disorders and, in the second place, in helping A. P. Brestkin to set up his physiological experiments, I directly witnessed the data being obtained.

A. P. Brestkin thoroughly checked the "endogenous-exogenous" theory of the genesis of decompression disorders developed by M. I. Yakobson, and in so doing, he proved its absolute untenability. A special series of experiments was undertaken to check the conclusions drawn by Ewald and Kobert about the lungs' permeability to air, on which Yakobson leaned in forming his conception. Repeating the experiments of Ewald and Kobert in an improved version, A. P. Brestkin established that incident to the

artificial respiration of animals under increased pulmonary pressure, at certain values of the latter after a time ruptures are formed in the pulmonary tissue, through which air penetrates into the vascular system. It turned out that the deep narcosis employed by the above-mentioned authors in their experiments, by weakening the tonus of the muscles of the thorax and smooth musculature of the bronchial tree and by reducing the elasticity of the lungs, to a considerable extent promoted the formation of ruptures of the pulmonary tissue under the indicated conditions. Healthy unanesthetized animals are able to bear breathing (in the present case, actively) under a rather considerable intrapulmonary overpressure without any lesions to the pulmonary tissue; this ability differs for different animals, being much greater in dogs than in rabbits. Hence, the facts show that the lungs, so long as they are whole, are impermeable to air bubbles. Also carried out by Brestkin were experiments in which rabbits that had been under increased pressure for a certain time had air injected into their gastrointestinal tract before decompression or had the communication of their lungs with the atmosphere restricted by constriction of the trachea. In both cases, according to Yakobson's theory, decompression disorders ought to have been induced to a considerable degree. But the experiments showed that there were no significant differences between the frequency and seriousness of decompression disorders in these rabbits and in the controls. Thus, Yakobson's main thesis, on which he built his theory -- namely, that the chief and indispensible cause of postdecompression gas formation in the organism is gas bubbles from the lungs and intestine getting into the blood -- proved to be erroneous. Also laid bare in the investigations by A.P. Brestkin was the untenability of Yakobson's assertions about the exceptional ability of the organism's tissues and media to retain nitrogen in the form of a supersaturated solution. Experiments on animals as well as with supersaturated gas solutions in different fluids in vitro concurred in showing that up to certain degrees of supersaturation, the formation of gas bubbles that are visible to the naked eye is absent; but when the maximum permissible degree of supersaturation is exceeded, bubble formation is always noted. It should be said that this circumstance does not in itself serve as a basis for rejecting the conception of Harvey and his coauthors about the role played in the process of postdecompression gas-bubble formation by gas nuclei that preexisted in the organism's media. Taking Harvey's position, it is possible to assume that incident to supersaturations that are below the maximum permissible supersaturation, the growth process of the gas nuclei is invisible to the naked eye, or that incident to certain degrees of supersaturation, the gas nuclei are stimulated to rapid growth. These assumptions, however, can in no way upset the fundamental fact that gas bubbles that are visible to the naked eye do not appear in the organism's media incident to subliminal degrees of supersaturation but regularly arise after a certain threshold of supersaturation is exceeded. Everything that has just been said already goes beyond the bounds of Yakobson's "endogenous-exogenous" theory, the untenability of which became clear and unmistakeable after these investigations of Brestkin's.

A study of postdecompression gas formation in different tissues and media of the organism undertaken by A. P. Brestkin yielded very important results. In experiments involving the complete saturation of rabbits with nitrogen under different pressures, killing of the animals in a chamber, subsequent decompression and autopsy of their corpses, the author showed that different tissues possess different abilities to retain nitrogen in the state of a supersaturated solution. It is fatty tissues that most stably retain the nitrogen with which they are supersaturated; gas formation takes place in them, according to Brestkin's data, at a coefficient of supersaturation (ratio of the pressure of the nitrogen in the tissues to the surrounding presure) equal to 2.8 - 3.2, or even in excess of these figures. In the blood, bubble formation takes place at considerably lesser degrees of supersaturation (coefficient from 2.4 to 2.8); finally, in the lymph, the fluid of the interfascial spaces and the synovial fluid, gas formation takes place still more readily -- at a coefficient of supersaturation equal to 2.24 - 2.4. These data of the author's are in conformity with the results of his experiments on the stability of supersaturated solutions of inert gases in fluids in vitro. These experiments showed that the stability of such solutions is the greater, the higher the solubility of the gas in the given fluid and the greater the latter's viscosity. For supersaturated solutions of nitrogen in the tissues, as Brestkin showed, the same relation holds good: fatty tissue, which possesses the highest viscosity and which dissolves nitrogen in the greatest quantities as compared with other tissues, /55 retains it in the supersaturated state most stably; but the blood, being considerably less viscous and dissolving nitrogen less well, shows a much lesser stability of its supersaturated solutions. These investigations once again raise the question of the role of the blood and the significance of the fatty tissues in the development of caisson phenomena. It had been known for a long time that, on the one hand, an abundance of fat in the organism is very conducive to the development of decompression disorders and, on the other, that incident to the autopsy of animals and human beings that have succumbed to caisson phenomena, the greatest amount of free gas bubbles is found not in the fatty tissue or other tissues of the organism, but in the venous blood. From fatty tissues supersaturated with an indifferent gas, the latter diffuses into the blood flowing through them and here can already form free bubbles at degrees of supersaturation at which no gas will yet be formed in the fatty tissue itself. Practically speaking, such relations, when, as the result of decompression, the coefficient of maximum permissible supersaturation is to some extent exceeded for the blood, but has not yet been reached for the fatty tissues, are more probable. Thus, in the light of the foregoing, it becomes clear that incident to decompression, the fatty tissues assume the role of a capacious depository of inert gases, putting up for a long time with the supersaturation of the blood flowing out of them that is so dangerous

from the standpoint of gas formation. Unfortunately, A. P. Brestkin's data about the different abilities of the organism's different tissues and media to retain inert gases in the state of a supersaturated solution, as well as the above-mentioned results of his studies aimed at elucidating the conditions under which gas bubbles are formed in supersaturated solutions, are not being made use of in the literature either sufficiently or in conformity with their great significance as matters of principle.[7] Very often researchers with equal reason go on discussing the possibility of gas-bubble formation in the blood and tissues after decompression, not infrequently getting bogged down in incorrect hypotheses about gas first being formed in the fatty tissue. As a result, the question of where in the organism postdecompression gas bubbles first and foremost are formed does not receive a clear and correct explanation. With this primary question in such a sorry state, it is quite natural that ideas about the organism's reaction in response /56 to postdecompression gas formation and about the intimate mechanisms of development of decompression symptoms should not be clear, either. The data in the literature bear witness to the fact that the pathogenesis of decompression disorders can be regarded as established only with regard to their most serious and fatal forms, when, incident to excessively high degrees of supersaturation of the organism, such intensive gas formation arises in the blood that it becomes impossible for the blood to circulate. But as far as all other manifestations of decompression disorders are concerned, be it a question of spinal afflictions, cerebral symptoms or the more frequently encountered symptoms, namely, the bends, itching of the skin, as well as general weakness and feelings of breakdown, the mechanism of development of these symptoms and conditions remains as unclear as before, or, in any event, not demonstrated.

It is significant that the interpretations of the mechanism of decompression disorders that exist in the literature deal with its individual manifestations and do not embrace the question as a whole. One might say that researchers endeavor to regard the pathogenesis of individual forms of "decompression sickness" as, for example, clinicians regard the pathogenesis of different forms of tuberculosis. But the crux of the matter is to elucidate the general mechanism of development of decompression disorders

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[7] This also applies to the later and very significant and solid study by A. P. Brestkin [1958], in which the author, in addition to the material he obtained in experiments on gas formation in animals and in supersaturated solutions in vitro, presents his detailed theoretical research concerning supersaturated solutions of gases in fluids and discusses the principal conditions that determine the genesis of stable gas bubbles in such solutions.

as a whole, to establish the general principles governing the organism's response to postdecompression gas formation of different degrees of intensity, to study the organism's reactions to this extraordinary factor and to determine the physiological significance of these reactions. In elucidating these questions, interpretation of the mechanism of development of the individual symptoms and different concrete manifestations of decompression disorders poses an incomparably easier problem. But especially conspicuous are the gaps in our understanding of the organism's physiological defense reactions in response to postdecompression gas formation. The data in the literature attest to the fact that not only has this question, so important from the theoretical as well as practical standpoints, not been subjected to experimental treatment; it has not even been raised by researchers.

The formulation and study of this question in our investigations, which will be the topic of discussion below, were predetermined by the whole complex of studies carried out under the direction of Professor M. P. Brestkin and the generalizations he derived regarding the organism's interaction with extraordinary factors. As has already been mentioned, under the direction of Professor M. P. Brestkin these studies were conducted on a broad front and concerned the effect on the organism of factors of a changed gaseous medium as well as other extraordinary factors, in particular overloading, rocking, shock waves and others. Apparent in one form or another in every instance of these studies was the organism's ability to resist extraordinary effects and the possibility of active physiological defense against their unfavorable influence. From this it was that Professor M. P. /57
Bresktin derived the general principles of the organism's interaction with extraordinary factors, principles concerning the organism's physiological reserves and their mobilization, which was the topic of the preceding chapter. These generalizations determined a single methodological approach to the study of different extraordinary effects on the organism. On this basis, in the study of decompression disorders, too, it was natural to take up the question of the organism's ability to resist their development. Such was the conceptual and factual premises for the investigations that constitute the content of our study.

PART 2

PHYSIOLOGICAL ANALYSIS OF DECOMPRESSION DISORDERS ARISING IN THE ORGANISM AFTER A STAY UNDER INCREASED PRESSURE

CHAPTER 4

SOME PROCEDURES OF STUDY OF DECOMPRESSION DISORDERS IN EXPERIMENTS ON ANIMALS

It will be expedient to preface the results of physiological /58
analysis of decompression disorders arising after a stay under increased pressure that are discussed in this part of the book with a description of some typical procedures that may prove useful in solving this complex problem. Such a description will, in the first place, enable the reader to get a better idea of the conditions under which the experimental data presented below were obtained and, in the second place, may be of use to persons who are about to enter upon research in this field.

Before describing these procedures themselves, it is necessary to say a few words about the most suitable animals to use for study of decompression disorders that arise after a stay under increased pressure. In this connection, there can be no doubt that the first place among all laboratory animals must be assigned to dogs. This is determined by, among others, the three following reasons.

In the first place, as compared with other laboratory animals, dogs are distinguished by their higher organization and the data obtained on them more closely characterize the physiological principles that are operative in the human organism. In the second place, it pays to use dogs in chronic experiments aimed at studying the decompression disturbances that arise after a stay under increased pressure because they display very clearly the joint symptoms of the bends type that are specific to such disturbances. Finally, the third advantage of dogs for the case in question is that while their resistance to decompression disturbances does exceed that of people, this difference is not so /59
great. Close to dogs in their resistance to decompression disorders are rabbits. In these animals, however, muscular and joint symptoms of the bends type are far from appearing so clearly. Moreover, rabbits are distinguished by their considerably lower general organization as compared with dogs and a relative weakness of two major vital systems: the cardiovascular and respiratory systems, which are of paramount significance incident to decompression disorders. The weakness of these systems, the instability of the basic parameters of their activity, the peculiarities of

the nervous regulation of heart action (weakly expressed vagotropic regulatory effects) -- all these circumstances, which are obviously connected with the low mobility of rabbits, tell negatively on the study of decompression disturbances in these animals and at times cause the results to be insufficiently stable. But the closeness of rabbits to man and (to a somewhat lesser extent) to dogs in their general resistance to decompression disorders permits them to be positively used for analysis of some questions concerning the mechanism of development of decompression disorders.

Cats, which, as compared with rabbits, are very vigorous, mobile and viable animals possessing great physiological reserves of the cardiovascular system and respiration, are generally not suitable for the study of decompression disorders because, in the first place, these animals, in virtue of the above-mentioned characteristics, prove to be much more resistant to decompression effects and, in the second place, they rarely display in a clear form the symptoms of the bends that are typical of decompression disturbances.

As far as small laboratory animals are concerned, it is obvious that guinea pigs can be used to advantage for the study of decompression disorders because, according to data in the literature, they are not far from man in their resistance to decompression disturbances and display relatively clearly the local signs of these disturbances [Bukharin, 1958].

In our experiments, guinea pigs were not used and I have no personal impressions about the pattern of development of decompression disorders in them.

As far as rats and mice are concerned, their resistance to decompression disorders is incomparably higher than that of people; experiment has shown that it is difficult to count on studying in these animals the dynamics of decompression disorders that are typical of man. Symptoms involving the extremities (the analog of the bends in man) do not appear constantly in these animals and when they do appear, it is most often under the impending threat of serious overall disturbances connected with aeroembolism and disorders of the blood circulation. Rats and mice can be /60
used chiefly in experiments with strong decompression effects designed with an eye to the survival-rate criterion.

These considerations on the choice of animals for the study of decompression disorders that we have just stated will receive additional substantiation in subsequent chapters where the outward picture and the inward mechanisms of decompression disturbances will be described.

Now let us go over to a description of some procedures which, taking into account the experience acquired in using them in our

investigations, there is cause to believe may prove useful in the hands of other experimenters working in this field. First of all, it is necessary to touch upon those frequently essential experimental conditions under which artifical mixtures of one sort or another must be administered under changed pressure conditions to an animal placed in the chamber and the experimenter is obliged to be close to the animal during the action of the increased pressure or during decompression (for the purpose of different additional interventions or the recording of physiological indices) but must not be subjected to the effect of the gaseous mixtures used for the animal.

For such experiments, which are as a rule conducted on dogs, a system of so-called continuous operation can be recommended, a diagram of which is shown in Fig. 1.[1]

The necessary gaseous mixture from a cylinder is supplied through a connecting pipe to a Douglas bag located inside the chamber, and the animal inhales this mixture from the bag through valves. The animal exhales through a hydraulic gas meter (to meter pulmonary ventilation) into a second bag located on the other side of the valves. This bag, too, is attached to a connecting pipe that passes through the chamber wall; an external valve permits it to be deflated, whenever necessary. Thus, the animal breathes the required gaseous mixture out of an isolated system of continuous operation, while the pressure in the chamber is raised by means of air. The chamber can be fitted out with the necessary equipment and instruments. The experimenter can be either beside the animal throughout the entire experiment or enter the chamber at the required times and emerge from it through an air lock.

In all cases, of course, all medical and technological requirements must be observed that ensure the safety of such actions on the experimenter's part. A system that is similar except for being provided with two Douglas bags at its input section can be used for an alternating supply of different gaseous mixtures for the animal to breathe. At low pressures that do /61
not threaten man with any unfavorable effects, alternation of the mixtures can be performed by the experimenter inside the chamber. But the alternate use of different mixtures is of primary interest in the case of high pressures, when the experimenter is obliged to remain outside the chamber. Here some special apparatus becomes necessary to permit the experimenter outside the chamber to supply the animal at will with one or

[1] Beginning with 1939, such systems have been extensively employed in a number of studies of the toxic effect of oxygen [Zhironkin, 1939, 1955; Panin, 1958; Sorokin, 1958; Gramenitskiy and Sorokin, 1964, etc.].

another gaseous mixture for breathing, alternating this supply in conformity with his intentions. Such an apparatus was designed and built by myself together with N. N. Feklistov. It must be said that the problem raised could undoubtedly have been solved by using sufficiently powerful electromagnetic devices. But the introduction into a high pressure chamber of a more or less powerful electric current, even in the case of a DC circuit and the absence of contacts that could cause a spark, is undesirable; therefore we built the apparatus in question on the basis of a mechanical principle. A diagram of this apparatus is shown in Fig. 2 and an outer view of its individual parts, in Fig. 3. We based ourselves on the principle of operation of the hydraulic braking system of an automobile, and the entire apparatus was built from the appropriate components of a Moskvich 401 automobile. To the outside of the chamber was attached the main braking cylinder (a), and its piston was connected to a lever (b) equipped with a stopper (c). The main braking cylinder was connected by means of a copper pipe (d) to a special three-way cock (e). From this cock two copper pipes led to connecting pipes that passed through the chamber wall (f_1 and f_2). To the connecting pipes inside the chamber were attached flexible braking hoses (g_1 and g_2), each of which led up to a small (wheel) braking cylinder (h_1 and h_2). These cylinders were fastened to a wooden foundation with their pistons up. The pistons were connected to levers (i_1 and i_2) which pinched wide elastic rubber pipes connected to the inspiratory valve through a T-pipe (j). By means of thin elastic rubber tubes (k_1 and k_2), the long arms of the levers were drawn up to the bar made fast above them. The ratio of the arms was taken to be 3 to 1; their length was equal to 18 and 6 cm, respectively.

The entire system of cylinders and pipes was filled with hydraulic braking fluid from a tank (ℓ), after which, in order to remove air bubbles from the system, it was "bled" in accordance with all the rules that are observed when running the automobile.

In using the described apparatus, it is necessary to connect the main cylinder with one of the small cylinders inside the chamber by means of an appropriate turn of the three-way cock, /62 clamp down on the lever (b) and stop it. In so doing, the corresponding rubber tube, as is shown in Fig. 2, will be pinched. At the proper time we must release the lever (in which case the tube becomes unclamped), turn the three-way cock by 180°, clamp down on the lever again and stop it. In so doing, the other pipe will be pinched. The power developed by such a /63 system is very great and easily opposes the pressure exerted by the compressed air on the pistons in the small-cylinder chamber. In any case, we used the described apparatus at a chamber pressure of 10 psig, and it worked reliably.

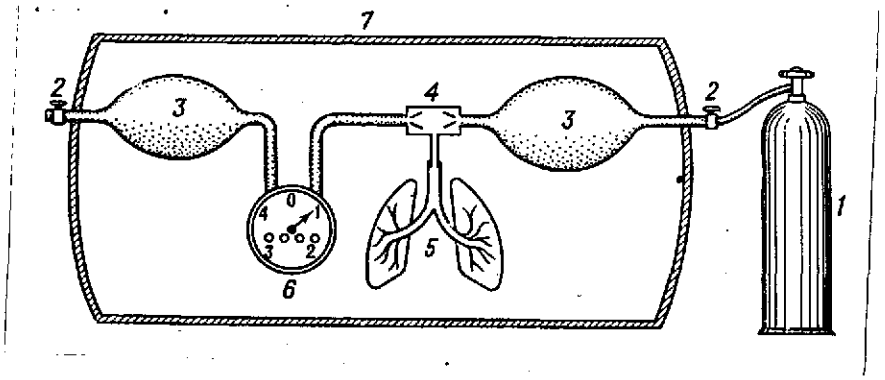

Fig. 1. Diagram showing isolated system of continuous operation for breathing different gaseous mixtures under increased pressure conditions.

1. Cylinder with gaseous mixture; 2. Inlet and outlet valves; 3. Douglas bags; 4. Inspiratory-expiratory valve; 5. Animal's lungs; 6. Gas meter; 7. Increased-pressure chamber.

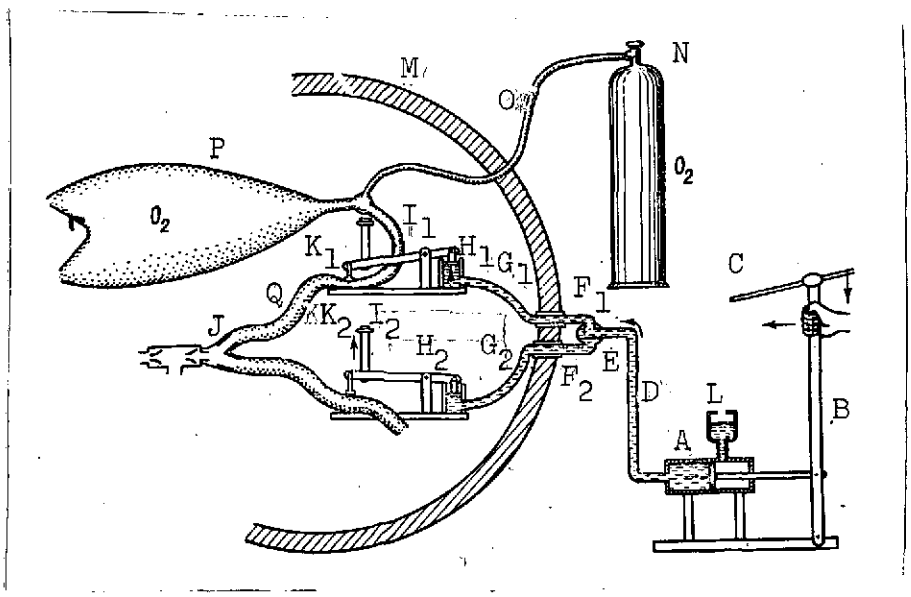

Fig. 2. Diagram showing apparatus for alternate supply of respiratory gaseous mixtures to animals under increased pressure. Cf. text.

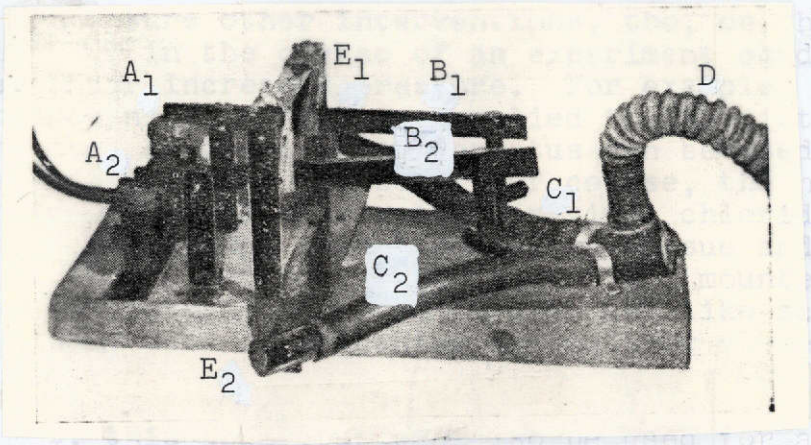

Fig. 3. Part of apparatus (outside chamber) for alternate supply of respiratory gaseous mixtures to animals.

$a_{1,\ 2}$ -- small braking cylinders; $b_{1,\ 2}$ -- levers that alternately pinch the respiratory hoses; $c_{1,\ 2}$ -- respiratory hoses; d -- common respiratory hose going to valve box and animal's lungs; $e_{1,\ 2}$ -- pipes for connection of Douglas bag.

It seems to us that such an apparatus, when suitably modified, can ensure other interventions, too, on the part of the experimenter in the course of an experiment conducted in the chamber under increased pressure. For example, if we see to it that mechanical effort is supplied to the piston of a suitably mounted syringe, this apparatus can be used to administer drugs to an animal under pressure. Of course, the needle connected to the syringe by means of a rubber or vinyl chloride tube must be introduced beforehand into a vessel or tissue and fixed there. Moreover, if the cylinders in the chamber are mounted in such a manner that the levers connected to them act like scissors, this apparatus can be used for pinching and cutting vessels or for neurotomies.

Finally, this apparatus can also be used for applying to the animal different mechanical stimulations (pinching, squeezing, touching, pricking). In so doing, it will probably be advantageous to use additional fluid-filled systems like the well-known /64 tactile stimulation devices and by means of them transmit mechanical forces to the animal. In this case, by limiting the

amplitude of the motions of the levers in the chamber, it will be possible to make sure that repeated stimulations have the same strength.

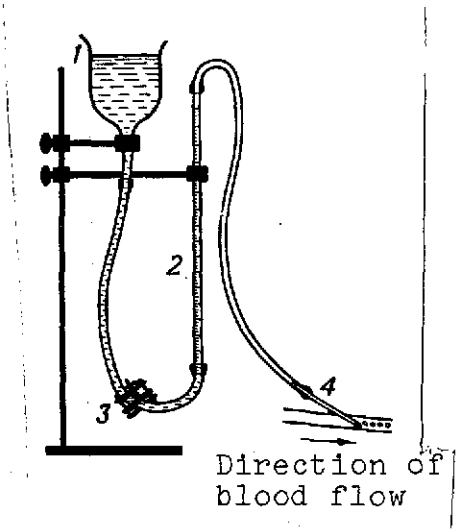

Fig. 4. Diagram showing system for intravenous gas administration. 1. Glass funnel with a capacity of 3ℓ; 2. 50 mℓ buret; 3. screw clamp; 4. needle to be introduced into vein.

In the course of our experimental investigations it became clear that a very useful and promising procedure for analysis of decompression disorders is artificial aeroembolism deliberately induced in the animals by the experimenter. It is a question of introducing a dose of air, or other gaseous mixtures and gases, into the vascular system. To this end we used two different systems that can be recommended for such investigations. The simpler system (Fig. 4) consists of a large 3-ℓ funnel from a gas meter and a 50-mℓ gas buret interconnected by means of a rubber tube with a screw clamp that regulates its lumen. This system of communicating vessels is filled with water. Onto the upper extremity of the buret is slipped a rubber tube which, during the gas administration, is connected to the cannula or needle fixed in the vein. Before the experiment tube and buret are filled with one or another gas down to zero pressure, the screw clamp is screwed tight and the funnel with water is attached 20 cm above the buret. The system is connected to the venous cannula (needle). On opening the screw clamp, the gas under the fluid head begins to enter the vein. This system meets the requirements of experiments on cats and rabbits, to which air and gases can be administered in comparatively small amounts. But in experiments with dogs, to which it is necessary to administer air and gases in considerably greater doses, such a system proves to be unsatisfactory: but when this buret is replaced by a longer and larger-capacity one, as the gas is displaced by the water, the fluid head decreases, and consequently so does the flow rate of the gas into the vein. Therefore, in experiments on dogs, it is expedient to use another system, more complicated, but also more efficient (Figs. 5, 6). A glass sphere (a) with two outlets is connected by its lower part to a graduated 100-mℓ buret (b) by means of rubber tubes, and by its lower part, to the bag of an

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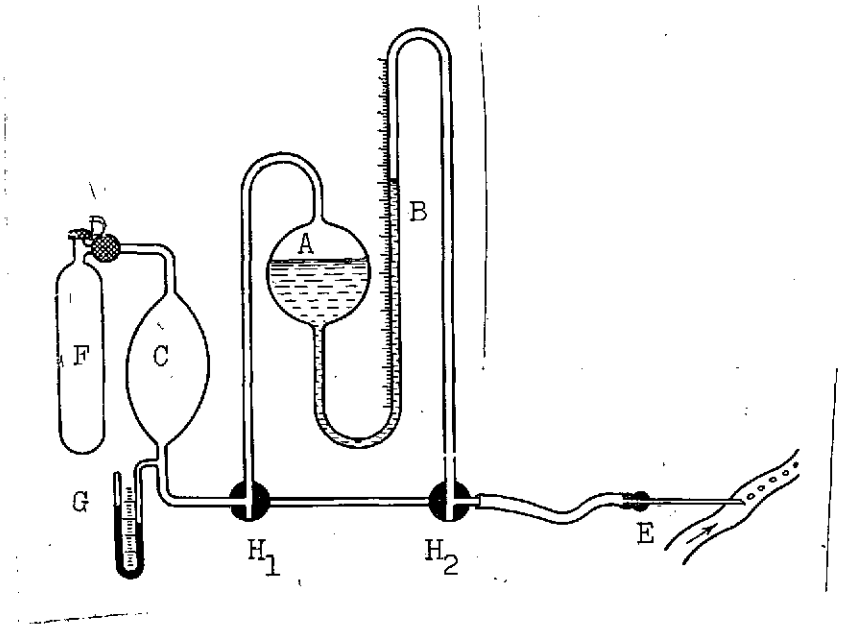

Fig. 5. Diagram showing improved system for intravenous gas administration. a -- glass sphere with a capacity of 3ℓ; b -- 100-mℓ buret; c -- bag of ISAM-48 apparatus; d -- reducer; e -- needle to be introduced into vein; f -- cylinder of ISAM-48 apparatus; g -- mercury manometer; $h_{1,\ 2}$ -- three-way cocks.

ISAM-48 apparatus (c) by means of a three-way cock. Through the reducer (d) attached to the cylinder, air (oxygen, helium) is fed to the bag of this apparatus, and there an overpressure is created whose magnitude is shown by a mercury manometer. Attached to the bag in parallel with the manometer is a mercury valve and vertically fastened to the screw support is a glass tube submerged by its free lower extremity into a tall beaker with mercury (not shown in the figure). In order to prevent the mercury from splashing the beaker is plugged at the top with a rubber plug having in its center an orifice somewhat greater in size than the diameter of the glass tube. The intensity of the pressure in the bag is determined by the depth of immersion of the tube in the mercury and is smoothly regulated by the screw device on the support. The excess of air (or any gas) that is continuously entering the bag emerges through the glass tube, bubbling outwards through the mercury, owing to which one and the same constant overpressure is maintained in the system during its operation. Under the action of this pressure, the water filling the glass sphere passes over to the buret, and the air

or gas from the buret, into the animal's vein. From beginning to end, the water head and, consequently, the flow rate of the /66
air (gas) into the blood, too, remains unchanged. Another advantage of this system is that by appropriately turning the three-way cocks, it is possible to force the water into the opposite direction -- from the buret into the sphere and, thus, rapidly prepare the system for the alternate administration of /67
air or any gas.

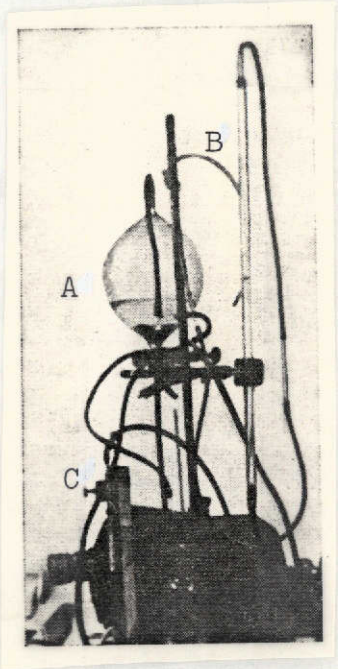

Fig. 6. Outer view of system for intravenous gas administration. a -- glass sphere; b -- buret; c -- mercury manometer.

In those cases where the crux of the experiment requires the air to be administered to the animals (in particular rabbits) in very small amounts, it is easiest to inject it from a well-ground tuberculin syringe.

In this kind of chronic experiment, in studying decompression disorders that arise after a stay under increased pressure, as well as in investigating the phenomena of artificial aeroembolism, it becomes imperative to be able to record the animal's (using no anesthesia of course) different physiological indices, especially those concerning cardiovascular activity and respiration. Experience acquired in experiments conducted together with V. I. Arsen'yeva and K. S. Yurova entitles me to make certain recommendations about how to organize such experiments. Again it is dogs that are the irreplaceable and, probably, only suitable animals for such experiments. But the earnest of success in such experiments is the experimenter's skill in dealing with dogs and his patience during the long process of systematically accustoming the animals to the experimental conditions. In our experiments, dogs were trained to lie on their side in a specially made cradle and to breathe through a mask.

A cloth vest with straps for immobilization was put on the dogs before they were laid in the cradle. The training process lasted 2.5 - 3 months with daily (except days off)

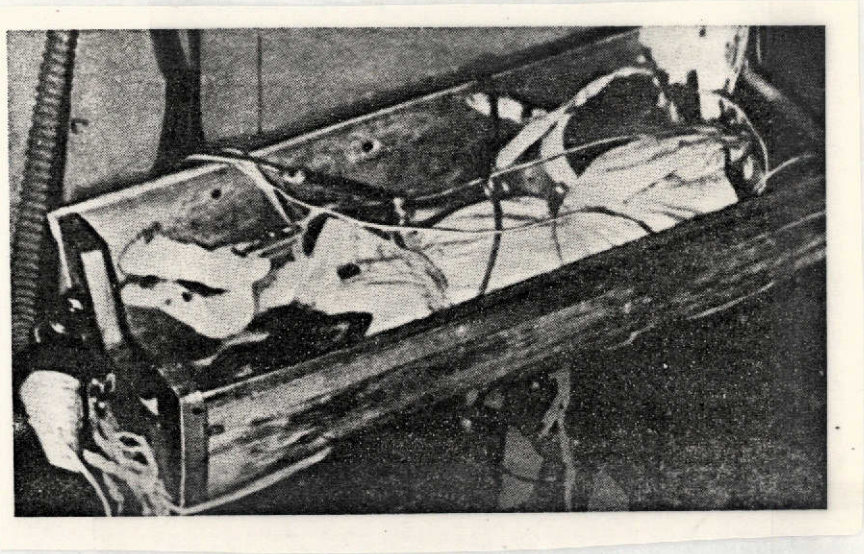

Fig. 7. Straightjacketed dog lying maskless on its left side in cradle.

confinement of the dogs in the cradle and, of course, with observance of the necessary general rules of animal training (positive reinforcements with food after every other session, gradual lengthening of the periods of confinement in the cradle, etc.). It must be said that for all this preliminary labor, the experimenter is amply rewarded in setting up the experiments. In the course of the experiments (in our case, after decompression from under increased pressure and incident to intravenous administrations of air), the dogs lie quite tranquilly in the cradle for 1.5 - 2 h. Immobilization proves to be necessary only as a precautionary measure in the case where general motor excitation arises under the action of especially strong effects. In our experiments, such cases were the result of especially painful decompression symptoms or the maximum permissible doses of injected air. Apart from such situations, the experiments proceed quite smoothly. Before the start of work, we operated on the dogs: their right carotid arteries were exposed in skin flaps. In experiments with carbon pickups placed on the artery located in the flap as well as on the thorax, the pulse and respiration were recorded on an MPO-2 oscillograph; at the same time, the volume of pulmonary ventilation was tape- /68
recorded with the aid of a hydraulic gas meter with electric contacts. In addition to these indices, we took the electrocardiogram in three standard leads, using needle-shaped electrodes. The dogs kept quite still during the introduction of these electrodes as well as the puncture of their veins inasmuch as

they had grown accustomed to having their skin pricked with a needle early in the preliminary period.

Figs. 7 and 8 show a dog in the course of such experiments, and Fig. 9 shows a sample of the physiological indices recorded in one such case. To be sure, the possibility of setting up similar experiments does not in principle constitute something new for physiologists. I have dwelt on their description in the desire to stress the acceptability and advantage of this type of experiment for the study of decompression disorders and the principles governing the development of decompresion and artificial aeroembolism . To have the opportunity of recording a good number of physiological indices in a normal unanesthetized dog and at the same time watch the development of the symptoms of the bends (which dogs lying in the cradle display clearly, lifting the affected paw) is of great importance for the analysis of the principles governing the development of decompresion disorders, and in order to create such an opportunity it is unconditionally worth putting in the considerable labor that is needed to prepare experiments of this kind.

Our crucial experiments on dogs were conducted in a large, double-compartment, increased-pressure chamber, in which a kymographic device was installed. Morphine-urethane or morphine-hexenal narcosis was used; a tracheotomy was performed, one of the femoral arteries was prepared and the gastric fistula was filled in order to prevent the phenomena of meteorism incident to decompression from under increased pressure. Experiments showed that without introducing an open cannula into the stomach, meteorism incident to decompression as a rule proved to be so pronounced in anesthetized dogs that it acutely disturbed respiration and the activity of the cardiovascular system. In experiments on the kymograph, the blood pressure in the femoral artery was recorded in the usual way, the respiratory movements of the thorax were recorded by means of mechanical transmission and, as in the chronic experiments, the volume of the pulmonary ventilation was recorded. During the development of decompresion disturbances, the changes in the enumerated indices were compared with the intensity of formation of gas bubbles in the blood. To this end we kept an eye on various easily accessible vessels that had been prepared beforehand (jugular vein, femoral arteries and the veins and vessels of the subcutaneous cellular tissue of the stomach) and used special gas traps that will be described below.

In some of the chronic and crucial experiments, the change in the gaseous composition of the arterial blood was studied in the dogs after decompression as well as after intravenous administration of air. Blood was taken from the femoral artery (in the chronic experiments, in dogs placed in a stall) by means of the customary apparatus filled with mineral oil with an admixture of anticoagulant. The content of gases in the blood was determined on a Van Slyke apparatus. /69 /70

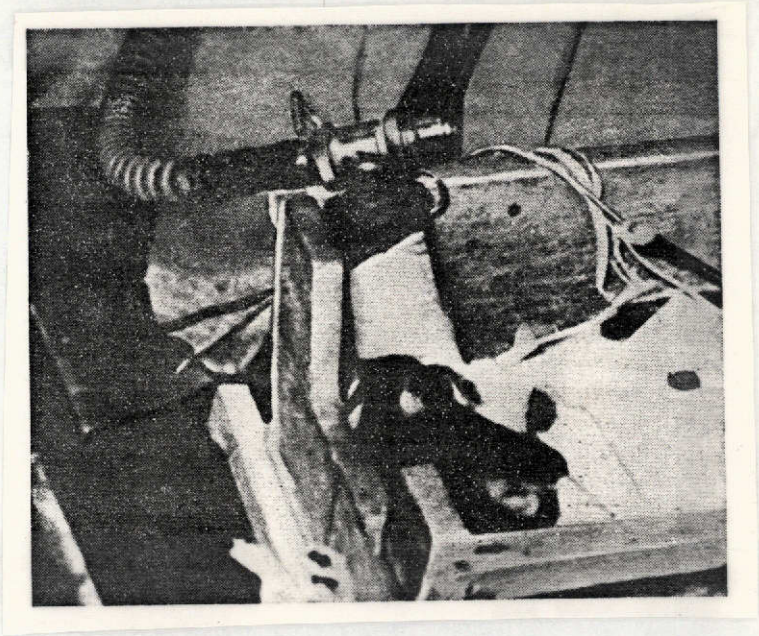

Fig. 8. Dog breathing through mask

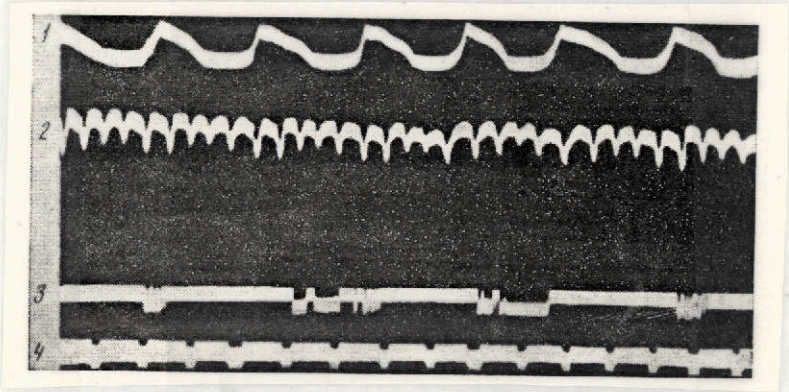

Fig. 9. Sample of physiological indices recorded in dogs in chronic experiments.

1. Respiration; 2. Pulse; 3. Pulmonary ventilation (marks in 200 mℓ); 4. Time, 1 sec.

The investigations on cats and rabbits were also conducted either in the form of chronic experiments, in which the animals were in a free state and under continuous observation, or in the form of crucial experiments, when, after appropriate vivisectional preparation, the blood pressure and respiration were

recorded on the kymograph incident to simultaneous checking (in the experiments with decompression) for the formation of gas bubbles in the blood.

In conducting crucial experiments on cats, we used urethane or hexenal narcosis. Most of the crucial experiments on rabbits were conducted without narcosis; here, incident to vivisectional interventions, we used novocaine.

It behooves me to dwell on methods of procedure and equipment employed in checking for the formation of gas bubbles in the blood of animals after their stay under increased pressure and decompression. Gas formation in the blood was studied only in crucial experiments inasmuch as no methods have yet been worked out for recording the presence of gas bubbles in an intact organism while carrying out the investigations under consideration.[2] In connection with the difficulty of determining the presence of gas bubbles in the blood of an intact organism, let us note the expediency of using in chronic experiments so simple a procedure as auscultation of heart sounds in the animals.

In studies by earlier diving physicians we find indications that during the development of decompression disturbances in divers, incident to heart auscultation crepitant noises were often detected, which were undoubtedly connected with the presence of gas bubbles in the heart cavities. Cases are even described where these noises were audible at a distance, especially interesting being the absence in a number of cases of any threatening general symptoms in the casualty at the time these noises occurred. Auscultating heart sounds in animals in chronic experiments, after serious decompression effects we often noted a characteristic gaseous crepitation, which was sometimes not yet accompanied by any acute functional disturbances.

As far as the intravenous administration of air is concerned, in these cases crepitation incident to contractions of the heart are very often audible to everybody around the experimental animal; the latter nevertheless displays no serious disorders /71 (unless we count intense panting) and soon learns to cope with the given effect. As is directly shown by experiments involving administration of air, crepitant noises in the heart arise only when a very considerable number of gas bubbles are present in the blood. Incident to decompression disturbances, these noises bear witness to an advanced stage of gas formation and appear for the

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[2] To this end foreign researchers have in recent years been using ultrasonic equipment; we had the opportunity to use such equipment not so long ago when studying altitude decompression disorders, about which more will be said in the appropriate section.

most part against a background of already pronouncedly developed functional disorders. Nevertheless, this symptom should not be ignored. It undoubtedly points to an abundance of gas bubbles in the blood, which it is important to ascertain also in those cases where pronounced decompression disorders are present, and sometimes it precedes serious functional disturbances, in which case it is particularly valuable.

In chronic experiments, we determined the appearance of gas bubbles and the intensity of their formation in the blood by direct observation of vessels as well as by special gas traps. For observation in experiments on dogs, the jugular and femoral veins were usually isolated and, in larger or smaller sections, the skin was separated together with the subcutaneous cellular tissue on the stomach and thorax. Acceptable for observation here proved to be the fine veins and arteries of the subcutaneous cellular tissue as well as the fine veins and arteries of the external wall of the stomach and thorax. Turning back the separated section of skin with its subcutaneous cellular tissue, it is convenient to inspect these as well as other vessels. In the breaks between observations, the separated skin can be returned to its place, whereby dessication of the tissues and their cooling are prevented; in a word, it is possible to maintain the conditions that are normal for the vessels and blood circulation and avoid side effects on the formation of gas bubbles in the blood. Observation of the fine vessels is of interest because the blood flow in them is much slower than in the main vessels, and their walls are much thinner; owing to this, gas bubbles can be noted (not only in the veins, but also in the arteries) that are at the limits of visibility for the naked eye. We confined ourselves to observation of gas bubbles in the above-mentioned vessels in those experiments whose main task was to study the reactions of the respiration and of the cardiovascular system incident to decompression disturbances and to compare these reactions with the process of gas formation in the blood.

In other experiments conducted on rabbits for the special purpose of investigating gas formation in venous and arterial blood after decompression from under different degrees of pressure, we used massive vivisectional interventions in order to place under observation the greatest possible number of different vessels. In these experiments, which were carried out under narcosis, long stretches of the large femoral vessels, carotid arteries and external and internal jugular veins were prepared; on extensive /72 sections of the thorax and stomach, skin with subcutaneous cellular tissue was separated; the abdominal cavity was extensively cut open for observation of the mesenteral vessels; the abdominal aorta, the posterior vena cava and the splenic, renic and iliac vessels were separated from the surrounding tissues. The vessels were uninterruptedly inspected for the presence of gas bubbles in a certain sequence: as soon as all the vessels had been checked, the next inspection was begun in the same order, etc, until the

end of the observations. In a number of experiments, at different periods after decompression we ligated different vessels and determined the possibility of appearance or additional formation of gas bubbles in the arrested blood.

It should be noted that small, single gas bubbles can be seen in medium- and, a fortiori, large-diameter arteries only under this condition. Because of the high flow rate of the blood in the arteries and the considerable thickness of the arterial walls, without ligation only large gas bubbles and gas accumulations proved to be accessible to observation.

To observe the formation of gas bubbles in the blood of experimental rabbits, we used the following procedure. Through an incision in the abdominal wall of the fixed animal, the loop of the small intestine was extracted and bound with ligatures to a wire half-ring fastened in the vertical position above the animal. The intestinal loop and its mesentary together with their vessels turned out in this case to be extended into fans.

Beyond the extended mesentery, a frosted bulb was lit; to moisten the mesentery, a loosely twisted cotton torniquet wetted with physiological solution was applied to the wire ring from above. Above the intestinal loop, a vessel with warm physiological solution was placed. Through a rubber tube whose lumen was regulated by means of a screw clamp, physiological solution dripped onto the cotton, and from there onto the mesentery. The vessels of the mesentery were very clearly visible in the light passing through, and to observe the advent of gas bubbles in them posed no problem. We were able to place the animal prepared in this manner into the chamber in the immediate vicinity of the sight glass and observe the formation and evolution of gas bubbles in the vessels of the mesentery incident to decompression and recompression.

The researcher, if he is properly attentive, can get a lot out of visual observation of gas bubbles in the fine peripheral vessels. In addition to determination of the kinds and intensity of gas formation in the vessels of different regions, it makes it possible to establish the character of the movement of the gas bubbles as a function of their sizes and numbers, as well as to keep track of how voluminous gas accumulations behave and when they cause arrest of the flow of blood in one or another vessel; /73 in a word, it leaves quite a clear impression of the course that aeroembolic phenomena take in the peripheral arteries and veins. In observing fine vessels, however, we always have to do with comparatively small regions, and, if we detect no bubbles here, we cannot certify that there are none in other sections of the vascular network. In the same way, from the amount of gas bubbles in the fine vessels under our observation, we cannot definitely judge the intensity of aeroembolic phenomena in the organism as

a whole. To this end, as well as to establish the beginning of gas formation in the blood, it is necessary to turn to the large main vessels.

But to rely on visual observation in this case is not permissible: the first, single, fine, gas bubbles may pass unnoticed through a large vessel together with the rapidly moving blood; when, on the other hand, the bubbles are many and they are clearly visible, it is possible to determine their total quantity and judge of the intensity of gas formation only in a rough approximation.

For more accurate determination of the beginning of gas formation in the blood and the intensity of the aggregate of aeroembolic phenomena, we used special glass gas traps that we set up in the path of the large venous and arterial vessels. The idea of using such gas traps arose and received initial approbation in heuristic experiments set up in 1950 by A. P. Brestkin and myself for the purpose of determining gas formation in the blood of animals incident to a rarefaction effect corresponding to altitudes of 8 - 9 km.

The design and action of the gas traps are illustrated in Fig.10. The inflowing blood rises up the inner tube of the gas trap, enters its resevoir, drops down and goes out through the lower outlet tube. The gas bubbles in the blood float up to the upper part of the gas trap, where they are accumulated. Before being introduced into the vessel, the gas trap is filled with physiological solution. Shortly before the experiment, this fluid, even if sterile, must be boiled or subjected to vacuum evaporation in order to remove dissolved gases.

Heparin is administered to the animals 5 - 10 min before the gas traps are inserted into their vessels.

In our experiments on dogs, we used gas traps with differing capacities (from 50 - 2 mℓ) and somewhat differing shapes (Fig. 11).

The capacity and shape of the gas traps is of primary importance. The greater the capacity, the more the flow rate of the blood passing through the gas trap is slowed down. This circumstance has its positive and negative sides. When the blood moves through the gas trap more slowly, the best conditions are created for trapping the gas bubbles that were in the blood. /74
In this case, it is less probable that fine gas bubbles with little buoyancy will not succeed in rising to the surface and will be carried away by the blood flow into the outlet vessel. Moreover, incident to slow passage of the blood through the gas trap, it can be assumed that the formation of gas bubbles takes place at the time the blood is in the instrument. These circumstances are of particular significance for the large arteries, where the blood moves very rapidly. Nor must we forget that a voluminous

gas trap, whether it be introduced into an artery or a vein, will always considerably lengthen the time of circulation of the blood in that region to which the given vessels belong; this will inevitably tell on the rate of desaturation of the tissues from nitrogen, and so on the degree of supersaturation of the venous blood flowing out from them, too. As far as the shape of the gas traps is concerned, for one and the same capacity, narrower and taller traps must be preferred to wider and shorter ones inasmuch as the former make it possible to judge more accurately of the quantity of air that is being accumulated in the instrument after certain lengths of time. If the main purpose of the experiment is to detect the first moment of gas formation in the blood, it is expedient to use small gas traps with a narrowing in their upper part: in them, single, fine, gas bubbles can be more easily seen. Of considerable significance in the action of the gas trap is the length of the inner glass tube up which the blood rises. If the upper edge of the tube is situated very close to the blister of the gas trap, then, ceteris paribus, it becomes more probable that the gas bubbles will "get through." With a great distance between the upper edge of the inner tube and the blister of the gas trap, on the other hand, it becomes more probable that /75 the blood standing still at the top of the instrument will coagulate (which does happen, in spite of the use of heparin).

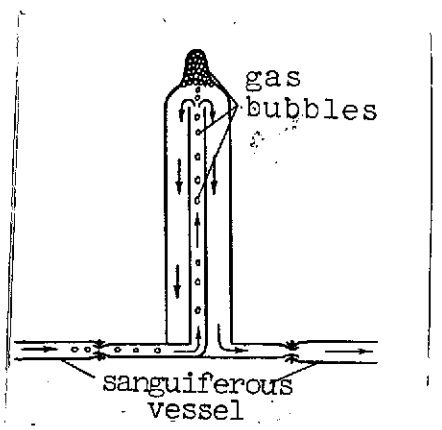

Fig. 10. Diagram of gas trap.

In the gas traps we used, this distance was about 2 cm for large-capacity traps and 0.5 cm for small-capacity traps. The gas traps in our experiments on dogs were introduced into the femoral vessels, the carotid artery and the external jugular vein. When simultaneously using an arterial and a venous gas trap in the corresponding regions, we always used contralateral vessels inasmuch as insertion of the gas traps on one side would lead to a twofold retardation of the blood circulation in the corresponding parts of the body.

In our experiments on cats and rabbits, we used small gas traps with a capacity of a few milliliters and introduced them for the most part into the cervical vessels.

My final word about the technique of using gas traps has to do with the method of connecting them to the ends of the transected vessel. We are convinced that only the smallest gas traps ought to be introduced by their inlet and outlet ends, which are shaped like cannulas, directly into the ends of the transected

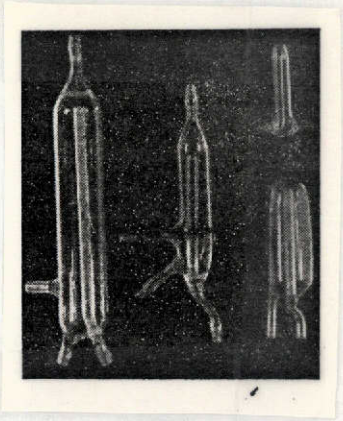

Fig. 11. Gas traps of different capacities and shapes.

vessels. Medium-sized and large gas traps can be more conveniently connected to the vessel through the intermediary of separate cannulas and short rubber tubes that can be bent in any desired way.

In experiments involving short exposure of the animals to pressure, the gas traps were introduced into the vessels before compression; but in experiments involving more prolonged exposure, they were introduced either immediately after decompression (the vessels were prepared beforehand) or not long before its

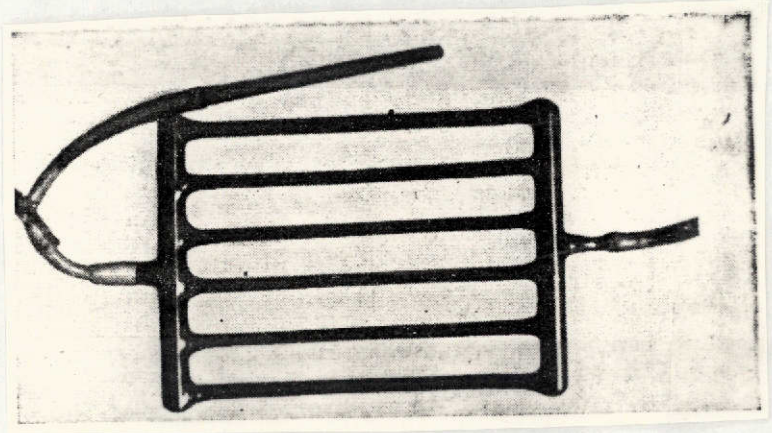

Fig. 12. Gas trap consisting of a number of parallel glass tubes (filled with blood).

beginning. In the latter case, the experimenter entered the /76
chamber through the airlock, either alone or with an assistant. In addition to the described gas traps, in the course of our work we tried out others that consisted of a number of parallel glass tubes (Fig. 12). They proved to be convenient for observing the first gas bubbles appearing in the blood. Getting into such a gas trap through the inlet tube, the blood at once splits into a number of parallel streams and sharply retards its motion. At the same time, the single fine bubbles arriving with the blood are immediately detected in the beginning part of one of the parallel tubes. By observing the subsequent slow passage of the blood along these tubes, we can establish whether new gas bubbles are being formed and whether the original bubbles are increasing in volume. Experiments showed that in a number of cases both take place. It should be noted that the use of gas traps has been subjected by some researchers -- partisans of Harvey's conception of the role of "gas seeds" in the process of post-decompression gas formation -- to rather harsh criticism based on the assumption that the insertion of a gas trap is connected with the introduction of such "gas seeds" into the vascular system and the blood from without. On these critical judgments and the significance of the slow blood flow in gas traps I shall dwell in the concluding part of the book, where the subject will be the recording of gas bubbles in the blood by this method and by the ultrasonic method.

But for now it makes sense to confine ourselves to what has been said about the technique and conditions of using gas traps. This will enable the reader, on the basis of the experimental material presented below, to form his own opinion about the extent to which the physiological shifts that characterize the development of decompression disorders in the organism correlate with the process of gas formation in the blood that can be recorded with the aid of gas traps, and the extent to which the latter are useful for studying the developmental mechanism of decompression disturbances.

CHAPTER 5

SYMPTOMS OF DECOMPRESSION DISORDERS IN DIFFERENT ANIMALS

Systematic study of all the outer manifestations of decom- /77
pression disorders has independent significance and will be of great assistance incident to physiological analysis of the mechanism of their development. To the results of observing animals after decompression from under increased pressure that are presented in the present chapter, we shall be obliged to return repeatedly later on in order to compare them with experimental data of a properly analytical character.

But it is necessary to describe the results of these observations before any other experimental data in order to make clear which symptoms we adopted as our criterion for the genesis of evident decompression disorders in experimental animals. Observations of animals after their exposure to increased pressure and decompression were conducted by myself and my colleagues K. S. Yurova, V. I. Arsen'yeva, N. Ya. Sidorov, A. A. Savich, A. A. Sagal in 380 experiments on 25 dogs, 42 experiments on 19 cats, 72 experiments on 57 rabbits and in experiments on 200 mice (V. A. Aver'yanov participated in the carrying out of these experiments).

Development of decompression disorders in dogs

Dogs, as is well-known to every researcher who uses them for the study of decompression disturbances, very frequently and clearly show specific decompression symptoms involving the extremities (symptoms of the bends). These symptoms are expressed in the fact that the dog stops using one or another extremity (to all appearances, because of painful sensations in it), bends it and holds it suspended. It is on the basis of such symptoms that it is generally accepted to judge of the genesis of decompression disturbances or, resorting to a widespread (in our opinion, incorrect) terminology, to speak of the outbreak of decompression sickness.

Steady observation of the animal from the moment of its emergence from the chamber, however, makes apparent a number of other, earlier and constant signs pointing to the development of decompression phenomena in the organism. Among such signs belong, in the first place, changes in respiration, the essence of which reduces to an increase in pulmonary ventilation. What is usually noted at first is an inconsiderable increase in respiratory rate; in a number of cases, the respiration frequency count shows no changes in rhythm, but upon observation of the dog, it is clearly apparent that the amplitude and the intensity of respiratory movements have increased, and there can be no doubt /78 that the intensity of pulmonary ventilation has increased. Later on, these small initial changes in breathing change into pronounced panting. The latter can take different courses. Sometimes this arises in the form of acute tachypnea, similar to heat panting; the dog opens its mouth and, protruding its tongue, begins to breathe extremely frequently, but superficially. In other cases, the panting is characterized by a more moderate increase in respiratory rate with its depth either unchanged or increased; the dog breathes with its mouth closed, but heavily and with an effort, as happens, for example, incident to an excess of carbon dioxide in the inspired air.

It should be noted that not seldom in the case of easily excitable, active dogs, panting of the tachypnea type arises every

time they get into the laboratory from the vivarium. Tachypnea in these cases is undoubtedly one of the manifestations of the animal's general excitation under the influence of the change in environment, movement, contact with the experimenter. Upon placing the animals in the chamber, tachypnea disappears, but, as a rule, upon emergence from the chamber after decompression, it immediately arises again and undoubtedly has the same basis. The sign of development of decompression disorders in such dogs is a cessation of tachypnea and a switching over to deepened and strained respiration with a closed mouth. Independently of the nature of the decompression panting, it is always noticeably increased, if the dog is compelled to move intensively; in this case, it is apparent that muscular work does not come easily to the animal.

Some or others of the described changes in respiration always and of necessity precede the development of symptoms involving the extremities, but what is especially to be stressed is that in a great number of experiments that do not terminate in symptoms of the bends, changes in respiration right up to pronounced panting also take place. Mostly, they arise 10 - 15 min after decompression, attain a maximum in 25 - 35 min, and then gradually ease up and disappear. It is significant that the maximum in the changes in respiration in experiments that terminated without joint symptoms coincides with those times when such symptoms developed in other experiments.

In the postdecompression period, the heart action of the dogs changes at the same time as their respiration. Evidence of this is the shift in the frequency and character of the pulse, which we palpated in most animals on the femoral artery, but in a few dogs with their carotid arteries brought out into skin folds, we palpated the pulse on a fold. The first changes in respiration are not seldom accompanied by an increase in pulse rate. Its extent varies considerably from dog to dog and experiment to experiment; for the most part, the increase in pulse rate amounts to 10 - 15 strokes per minute.

In a number of cases, the pulse rate remains unchanged, but upon palpation, the change in its character is clearly perceptible: it becomes stronger, sharper and more strained: in /79
clinical practice, such a pulse is called "pulsus altus;" its appearance in our experimental dogs obviously testified to an amplification of the contractions of the heart. Later on, with the accumulation of different functional shifts caused by decompression, in most of the dogs retardation of the pulse arises; the development of joint symptoms is as a rule preceded by bradycardia; when, however, these symptoms become very pronounced, the bradycardia is replaced by tachycardia, which we must assume arises in the given case as a result of painful impulses proceeding from the affected extremity.

It should be noted that in a special series of experiments, which will be discussed later on, the just-described respiratory and pulse changes after decompression were documentarily confirmed by graphically recording these indices on an oscillograph.

A great number of signs that precede joint symptoms and point to the development of decompression phenomena in the organism can be noted by observing the outer appearance, behavior and general condition of the dogs after decompression. Simultaneously with the first changes in respiration, the signs of general inhibition and depression appear in the animals. The dog loses its customary brisk appearance, becomes dull, inactive, lies down a lot and, if compelled to get up, tries to reassume a recumbent position. All of this is especially noticeable in frisky, active, sociable dogs. The dog's inhibition and depression also appear clearly in its reactions to external stimuli -- to every random noise and sudden change in the environment, to the experimenter's summons, his speech and different actions addressed to the dog, to food stimuli. Reflexes of the "what is this?" type as well as reactions to different positive stimuli proceeding from the experimenter (caresses, the call to go walking, etc), turn out to be suppressed to a greater or lesser extent; food reflexes are also slowed down -- often the dogs refuse their favorite food products: pieces of sausage, cheese, meat, sugar. All these signs, which are expressed to a greater or lesser extent, always precede the appearance of joint symptoms involving the extremities and, again, in a great number of cases, the consequences of decompression are even confined to these signs and the above-mentioned respiratory and pulse changes.

Sometimes added to all the enumerated signs that precede the symptoms of the bends are two others: the dog begins to scratch itself or, frequently knocking its teeth together "bites out" its hair and, as they say, "catches fleas." The scratching reflex arises now in the right, now in the left rear extremity and is addressed to different regions of the front part of the body. The "flea-catching" reflex also has its place of application at different points of the rear part of the body. /80 Both these signs are undoubtedly the analogs of the symptoms of post-decompression itching of the skin in man, but while the "itchy skin" symptom almost obligatorily precedes the onset of joint pains in people, the above-mentioned reflexes in dogs do not often, according to our data, put in an appearance.

As far as symptoms of the bends type themselves are concerned, these, too, usually arise not suddenly or at once, but develop from initial, hardly perceptible signs into pronounced disturbances of the position and function of the extremity. True enough, this development for the most part proceeds rather quickly. The first signs of the bends are that the dog begins to be sparing of one of its extremities. At first glance, it still stands quite normally on all four legs and does not limp in

walking; but, by intently observing the position of the extremities, we can see that the standing dog eases the load on one of them, scarcely perceptibly bends it at the joints and transfers the weight of the corresponding half of the body to the extremity on the other side. After that, the dog usually begins now and then to raise the affected extremity, but immediately replaces it on the floor every time (Fig. 13). Subsequently, the extremity is held in a raised position for ever longer periods, at first a slight limp develops in walking, and then a distinct lameness. Finally, a pronounced flection of the extremity sets in, the dog holds it suspended and no longer uses it at all in moving (Fig. 14).

In some cases, clear symptoms of the bends develop so rapidly that it is not possible to keep track of all of the just-described sequence of events; more often, however, a few minutes elapse between the first signs and a clear impairment of the function of the extremity. The degree of the symptoms in individual cases varies widely. If the animals are not subjected to therapeutic recompression immediately after the appearance of clear symptoms, the latter in the course of time most often steadily increase in strength: the flection of the extremity keeps increasing and attains extreme degrees, the animal falls into restlessness, begins to stamp and turn around in place on three legs; it turns it attention to the affected extremity all the time, the painful sensations become manifestly unendurable; the dog begins to howl and whine.

Fig. 13. Sedoy
21st minute after single-step decompression from under a pressure of 2.0 psig (exposure to pressure, 4 h). Against the background of general inhibition and panting that developed in the tenth minute after decompression, the first signs of the bends put in an appearance -- the dog lifted its left rear paw.

Fig. 14. Sedoy

23rd minute after decompression from under a pressure of 2.0 psig (same experiment). Panting has increased, a pronounced symptom of the bends has developed -- the dog holds its left rear extremity suspended in a bent position and makes no use of it either in standing or in moving.

In other, more rarely encountered cases, the development of the symptoms is confined to an inconsiderable flection of the extremity and a slight lameness in walking without progressing with the passage of time. In such cases, to judge by the behavior of the dog, the symptoms do not cause it any very great anxiety. The matter is confined to a moderate general depression of the animal and to the difficulties it experiences in moving. It should be noted that joint pains of moderate intensity in excitable dogs can temporarily abate and even disappear under the action of external stimuli. If the stillness of the laboratory /82 environment is suddenly disturbed and an attempt is made to excite the dog by one means or another, we can often see how the reviving dog as it were forgets about the sensations that cause and maintain the symptoms. For some short time the affected extremity assumes its usual position and begins to function as it should incident to the animal's movements and changes of position. The same thing happens in the case of active and easily excitable dogs, if they are led out of the laboratory into the courtyard and let loose. Not seldom such dogs with moderate symptoms of the bends developing in them make for the street door on three legs, but once at liberty, being released from their leash, they begin to run and romp, decidedly not showing any functional disturbances. To be sure, a change in the environment and the granting of liberty act in this manner only for a short while:

in a few minutes the former symptoms reappear under these new conditions. From the physiological point of view, these facts are quite understandable: under the action of sudden stimuli, the flectural reflexes of the affected extremity that are due to painful impulses are temporarily suppressed in accordance with the law of conditioned inhibition.

Sometimes the symptoms of the bends attain their limit intensity in a very short time: the dog, drawing in the extremity to its very body, hops, finding no place for itself, on three legs, squeals and whines. In this case, of course, no exogenous stimuli any longer relieve these acute painful symptoms. The photographs of Figs. 13 - 17 illustrate the development of different degrees of decompression symptoms in dogs.

In a number of cases, incident to the development of pronounced painful symptoms, the dog's actions point to a localization of the pain in the extremity. The dog keeps on turning its snout toward the affected paw and begins to lick it from time to time in a certain place. For the rear extremities, this is most often the region of the knee joint, less often of the hip joint and hocks and foot; for the front extremities, the region of the radiocarpal joint, less often of the elbow joint (there can be no question of the shoulder joint inasmuch as it is inaccessible to dogs). On observing such actions of the dog, we will notice that the region attracting its attention is not narrowly limited: the dog licks the hair and skin now immediately above the joint itself now in proximal or distal regions at a rather great distance from the joint.

Fig. 15. Ryzhik

19th minute after single-step decompression from under a pressure of 2.6 psig (exposure to pressure, 4 h). Panting and clear symptom of the bends involving the right front extremity.

Fig. 16. Valet

24th minute after single-step decompression from under a pressure of 2.4 psig (exposure to pressure, 6 h). Panting and clear symptom of the bends involving the left front extremity.

The localization of a pain in the extremity can sometimes be established by means of manual examination. To be sure, in the case of pronounced painful symptoms, this is difficult inasmuch as the dog reacts very strongly to any attempt to touch the affected extremity and, protecting it, often growls and snaps. In the case of more moderate symptoms this is easier, whereupon palpation does not yield much -- /84
as a rule, no bounded painful region can be established. On the other hand, examination of the joints by means of forced movement in a number of cases unmistakeably points to which of them is connected with the painful symptoms. The data of such examinations permit us to suppose that incident to the development of the symptoms of the bends involving the rear extremities, most often affected is the knee joint, more rarely the hip joint and the hock; in the front extremities, the painful symptoms are apparently most often connected with the radiocarpal joint and more rarely with the elbow and shoulder joints. The symptoms connected with the shoulder joint sometimes prove to be unusual: instead of flection, the extremity becomes extended and is deflected toward the side. The reservation must be made that the above conclusion about the frequency of affliction of the different joints is of a heuristic nature inasmuch as our data on this score are insufficiently complete. This is due not only to the fact that to determine the localization of pain from a dog's affected extremity is not always easy, but also to the fact that in view of the experimental conditions, it is often impossible to spend time on this. It is necessary to begin therapeutic recompression as soon as possible after the appearance of clear symptoms.

Experiments showed that in most dogs, decompression dis- /85
turbances of the bends type developed more frequently in the rear than in the front extremities. For individual dogs, the difference in the frequency of involvement of the rear and front extremities is not great; for the majority, however, it is very considerable. In some dogs, only the rear extremities suffered

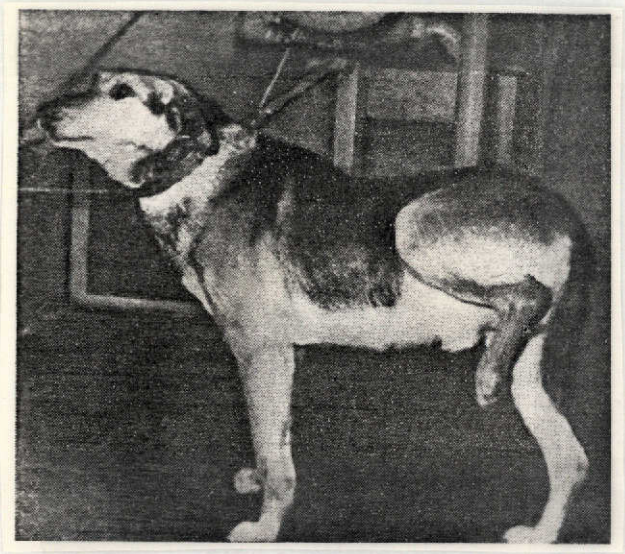

Fig. 17. Bogatyr'

7th minute after single-step decompression from under a pressure of 4.5 psig (exposure to pressure, 35 min). An acute symptom of the bends has suddenly developed: the left rear extremity in a state of extreme flection; signs of intense pain; the hitherto panting has come abruptly to an end, the ears are laid back, the dog whines.

in the experiments. Thus, the fact of their preferential involvement incident to the development of decompression disturbances in dogs is not open to doubt. In individual animals, as a rule, it is again one of the rear extremities that is distinguished by the frequency of its involvement. We became convinced that this circumstance is connected with the course of the experiments and the intensity of the painful symptoms. If particularly acute painful symptoms developed in one of the extremities of a dog in some experiment, then, in spite of the complete curative effect of recompression, this same extremity is again the one that will be most frequently affected in subsequent experiments. It is difficult to say whether the reason for this circumstance is some residual morphologic disturbances in the affected regions or whether conditioned-reflex mechanisms play a role here. Be that as it may, working with dogs for a long time creates the impression that in each one of them, when decompression disturbances of the bends type are repeatedly caused, one of the extremities suffers preferentially and behaves like a locus minoris resistentiae.

It is necessary to mention that in a number of experiments, when therapeutic recompression was begun not immediately after the appearance of the bends symptoms, the first extremity to suffer was joined by the other; the dog began to lift each of these extremities alternately and experienced obvious difficulty not only in walking, but also in standing in place.

As far as more serious decompression disturbances are concerned, to observe their development in dogs in chronic experiments was not, as has already been said, one of our aims. As soon as the bends symptoms appeared quite distinctly, we subjected the animals (for the most part immediately, but sometimes with a certain deliberate delay) to therapeutic recompression, which in

all cases, with rare single exceptions, yielded the full effect: after a certain exposure to increased pressure and subsequent gradual decompression according to special methods, the animals emerged from the chamber with no trace of functional disorders.[3] In some experiments, however, when the animals were subjected to the action of medium and high pressures, we chanced to encounter more serious decompression disturbances. In two cases, this was paralyses of the rear part of the body. They developed in the dogs after a prolonged stay under a pressure of 5 and 6 psig in the first stage of two-step decompression. The dogs were in the chamber, for which reason it was difficult to observe the /86 dynamics of how the paralyses developed. It was obvious that soon after rapid decompression to the first halt, the dogs began to pant intensely, and general depression and weakness appeared, compelling the animals to lie down. Owing to this, the bends symptoms, if indeed they did precede the paralyses, could not be noted, and therapeutic recompression was begun when it became evident that the rear extremities and rear part of the body of the animals were malfunctioning. In one dog, therapeutic recompression (the chamber permitted the pressure to be raised only to 10 psig) yielded no positive results: the paralyses proceeded to the front part of the body, severe respiratory disorders arose and the animal died. In the second dog, therapeutic recompression arrested the development of decompression disturbances at the stage of paralysis of the rear part of the body, but the paralysis proved to be persistent and irremediable: two weeks of observation of the animal showed that there was no hope for its recovery: the dog was destroyed.

In a number of experiments, we encountered the initial manifestations of pareses of the animal's rear extremities and rear part of the body. Moreover, in a few rarer cases, the paretic manifestations developed in one of the rear extremities against a background of prior bends symptoms. The active flection of the extremity that is characteristic of these symptoms was diminished, and the muscles of the extremity lost their usual tonus: the extremity hung lifelessly and was let down to the floor; the animal was in no condition either to rest on it, standing in place, or to make full use of it in walking. The true sign of developing paresis and of the concomitant disorders of sensitivity was the unnatural way of planting the extremity: the dog put it down on the floor not by the sole, but by the rear of the foot.

The development of paresis of one of the rear extremities always threatens to disturb its function persistently and

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[3] The different therapeutic decompression methods tested in the course of our investigations and their comparative efficiency will be discussed in Chapter 11.

irremediably, but (insofar as we could judge from the limited number of our observations) is not fraught with any great danger to the animal's life. Considerably more threatening and insidious are other cases, in which the manifestations of paresis with no prior bends symptoms take possession of the entire rear part of the dog's body. The outward signs of impending severe decompression disturbances in such cases are very skimpy. Often we succeed only in noting some wrong moves of the rear extremities as the animal moves and in just catching sight of isolated instances of slight and not very striking swaying of the rear part of the body. Before the rear extremities suddenly cease to support the dog; it collapses now on one side, now on the other: the rear part of the body at once proves to be paralyzed, and the animal's fate is decided in a matter of seconds: the least delay in therapeutic recompression threatens the dog with total paralysis and death. Of this we became convinced in conducting special experiments on two dogs involving rapid single-step decompression after an exposure to a pressure of 10 psig, and in the chronic experiments we were therefore particularly attentive to all /87
signs heralding such a course of events. Such signs even before the start of the slightest motor disorders in the rear part of the body are an intensification of panting and the appearance of particularly hard breathing, as well as a change in the dog's posture and outer appearance; standing, it begins as it were to hunch over, not seldom the hair along its entire back rises (Fig. 18), the general inhibition and depression are clearly intensified: dropping its head and breathing heavily, the dog stands motionless with an unusually curved back and remains indifferent to its surroundings. With the animal in such a state, the least motor disorders in the rear extremities and rear part of the body should be a signal for prompt therapeutic recompression.

In the course of the chronic experiments, we observed 12 of the just-described cases of incipient paresis of the rear part of the body in five dogs and six cases of paresis of one of the rear extremities in two dogs. Therapeutic recompression yielded the full effect in all these cases, with the exception of one, in which a slight paresis of a rear extremity persisted in the dog. The animal was temporarily barred from experiments and within a month and a half, the manifestations of paresis were completely liquidated.

Before describing the outward picture of decompression disturbances in other experimental animals, we deem it appropriate to note once again the special and incontestible advantages of /88
dogs as objects of physiological observations incident to studying decompression disorders. The exceptional value of dogs for physiological investigations conducted in the form of chronic experiments was shown in all its plenitude by I. P. Pavlov; it is generally known and requires no special proofs.

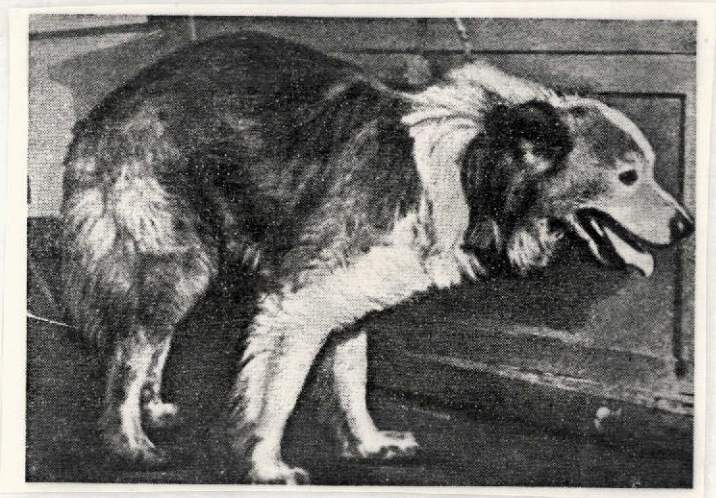

Fig. 18. Ryzhik

9th minute after two-step decompression from under a pressure of 6 psig (exposure to a pressure of 6 psig for 40 minutes and 2.1 psig for 30 minutes). The dog shows the signs heralding the development of paresis and paralysis of the rear part of the body: hunched back, unnatural position of both rear extremities, progressive depression and panting.

But chronic experiments connected with the effect of unusual external factors on the organism, and, in particular, with decompression and its consequences, show with particular clarity the inestimable qualities of dogs as experimental animals. Such experiments, moreover, compel our particular attention to a condition which is often overlooked, but upon which depends how fully the experimenter will be able to make use of the broad opportunities for physiological observations which the experimental dog presents him with.

It is a question, if one may say so, of the interrelations that are established between the experimenter and his experimental dog in the course of work. If the experimenter is apathetic and indifferent to the dog as to a "clever" live creature and, without trying to enter into contact with the dog, plays in each successive experiment the role of a dispassionate outside observer of the experimental animal, he will inevitably lose much of value in his observation and will not infrequently err in his appraisal of the general physiological condition of the organism. In every dog the course of evolution and historic development have instilled a deep and abiding instinct to have a human being for a master. If the experimenter knows how to satisfy this instinct, if he wins the dog's affection and becomes for it the person it

picks out from all others as its master, he will very much extend the range of opportunities of his observations. This can be achieved very easily: if the experimenter, without resorting to the assistance of a laboratory worker, will himself take the dog out of the vivarium, go walking with it before the experiment, display to the dog his affectionate relationship in different ways, not forgetting, of course, about supplementary feedings with tidbits at the proper times, the animal's enduring attachment to him will be ensured. In so doing, the experimenter will not only thoroughly study all of the animal's habits, all of the peculiarities of behavior that are characteristic of it on walks and in the laboratory and, consequently, will be able by the least changes in behavior to judge incipient general physiological shifts in the animal's organism; he will get a chance to associate with the animal, i.e., to appraise its reactions to his words and actions, which have acquired for the animal the significance of reinforcing and definite conditioned-reflex signals. All of this is especially important for experiments with numerous and repeated decompression effects inasmuch as the reactions of each individual dog that arise incident to the development of decompression phenomena in the organism, have their characteristic peculiarities. The latter concern not only different nuances of /89 the animal's behavior and his conditioned-reflex responses to the experimenter's treatment, but also the outward picture of the development of specific decompression symptoms involving the extremities. In some dogs, the approach of these symptoms is expressed in the persistent tendency to lick that extremity which is beginning to bother the animal; in others, on the other hand, it is expressed in increased mobility. Some dogs before the bends symptoms at once become inhibited and unsociable, while others at first make strenuous overtures to the experimenter, demanding his attention in return. Knowing all of these peculiarities of a dog's, it is possible to say beforehand whether the experiment will proceed without specific signs of decompression disorders, whether it will terminate in bends symptoms or whether more serious decompression disturbances will be impending.

Of the exceptional importance of all of the just-mentioned moments we became convinced for ourselves in conducting long investigations together with K. S. Yurova and V. I. Arsen'yeva. The dogs were used in experiments over a period of 5 - 6 years. All of the animals were subjected many times to decompression effects; many of the experiments were risky and posed a direct threat to the dogs' lives. But for all of the time we worked with them, out of 20 experimental animals used in chronic experiments, only three were lost, while we managed to keep the rest quite healthy and unharmed. For us, there can be no doubt that the outcome of the experiments and the fate of most of the dogs would have turned out different, if the experimenters had not had close contact with the animals and had not known all the details of the peculiarities of behavior of each dog and the characteristics of its reacting to the development of decompression

phenomena; this guaranteed against fatal delays in therapeutic recompression. Here it is necessary to note still another circumstance of interest for the general physiological characterization of the upper nervous activity of dogs. No matter how strong were the effects to which the dog was subjected in chronic experiments (altitude hypoxemia, oxygen poisoning, decompression disturbances), it does not connect these effects with the experimenter, if it behaves toward him as toward its master. The experimenter enters into the complex of conditions that precede and accompany these effects: when conditioned reflexes have been formed in the dog, it holds back when the experimenter leads it into the laboratory, offers resistance when he places it in the chamber: at the end of the experiment, the chamber frightens the dog still more, but toward the experimenter-master, it maintains its former positive relationship, and sometimes tends to fawn upon him even more than usual. The reinforced positive conditioned reflexes on which the dog's attachment to the experimenter is based, turn out to be so strong that they are not disturbed in these cases, even though here the experimenter and his actions constitute direct signals of the harmful effect.

Seldom noted in our work was the formation in the dogs of /90 negative conditioned reflexes to the furnishings of the experiment, in particular the chamber. This can be explained, firstly, by the fact that the onset of decompression disturbances is remote from the time of the effect itself (the stay under pressure and decompression) and, secondly, by the fact that experiments with pronounced decompression disturbances alternated with experiments in which only inconsiderable functional shifts took place (panting, the animal's inhibition) or in which there were no decompression symptoms at all. A clearly negative attitude of the animals toward the furnishings of the experiment (the chamber) appeared only at isolated moments of the investigations, when in some experiments the dogs developed pronounced decompression disturbances one after another. In experiments on three dogs, moreover, we observed the following very curious facts.

At the start of the experiment, the dog went to the chamber unwillingly or even offered active resistance, and it had to be placed in the chamber by force. But incident to the development of pronounced symptoms of the bends type, it rushed toward the chamber and, leaping on three paws and overcoming all difficulties, got into it by itself. Hence, the rapidly developing effect of therapeutic recompression, experienced many times, led to the formation of its conditioned reflex: the chamber, which at the start of the experiment was a clearly negative stimulus, changed, when the dogs developed painful symptoms, into a positive stimulus connected with the liquidation of pain. This fact points to the great sharpness and extensive resources of the upper nervous activity of dogs. It once again confirms their special value as objects of physiological observations incident to different

effects and, in particular, incident to the development of caisson phenomena.

Development of decompression disorders in cats, rabbits and small laboratory animals

In contrast to dogs, cats very seldom develop the bends, and it is more difficult to detect them. When cats are sitting or lying (and these are their most usual positions), functional disturbances in the extremities can go unnoticed. In order not to overlook them, it is necessary from time to time to compel the animal to move: then the appearance of symptoms of the bends can be immediately detected in limping or severe lameness. We observed the symptoms of the bends in cats only in four out of 29 experiments, which, according to other signs, were accompanied by very pronounced decompression disorders.

A very frequent decompression symptom in cats is signs of itching of the skin. These are expressed in terms of the cat beginning to scratch itself incessantly, and still more often "to bite out" its hair ("to catch fleas"). Sometimes this /91
symptom also appears in a weak form, while in other cases the cat continuously turns now to one, now to another part of the body and executes this reflex with such energy that it seems to be biting itself.

In spite of the fact that the signs of itching of the skin appear very often in cats after decompression, we became convinced that they cannot be adopted as a reliable criterion for the onset of decompression disorders.

These signs sometimes arise after the decompression of cats from under deliberately safe pressures, when the formation of gas bubbles in the organism is excluded. It is possible that they are caused in these cases by a change in temperature conditions incident to decompression or by some other side effects. Account ought to be taken of the fact that the reflexes of licking and "biting out" the hair are very frequent and usual in cats even under normal conditions: they are some of their constant, everyday actions and can obviously be easily caused by different, very insignificant stimulations of the cutaneous receptors. Further, these signs of itching of the skin in all those cases where they accompany the development of decompression phenomena, vary extremely in strength. If the described reflexes are executed continuously and intensively, if it is obvious that the animal literally finds no place for itself because of the unusual sensations proceeding from its skin, then the decompression nature of the symptoms becomes evident. If, on the other hand, the cat licks or "bites out" the hair in one or another spot only from time to time, then it becomes questionable to connect

these reactions with decompression phenomena. Nor, finally, must the signs of itching of the skin be regarded as a reliable criterion for decompression disturbances because, in a great number of cases, serious and sometimes life-threatening decompression disorders arise in the animals in the complete absence of these signs.

A constant and obligatory symptom of incipient decompression disorders in cats, as well as in dogs, is a change in breathing. As a rule, in all experiments terminating in pronounced decompression disorders, a light, not very striking, panting preceded their development by far. In a great number of cases, incident to less intense decompression effects, visible functional shifts were even confined merely to light panting and some inhibition of the animal. A steady increase in panting serves as a true sign of approaching decompression disturbances. The latter show themselves in cats in an impairment of coordination of movements and progressive depression of the animal. Its general condition deteriorates under one's very eyes: in the beginning, only an unsteadiness of gait is noted; then, usually, the rear extremities and the entire rear part of the body become weak; the cat loses the ability to move and sits rocking from /92
side to side, leaving its rear extremities in that often unnatural position they assumed when it became impossible for the animal to move. At the same time, its reactions to different distant stimuli become ever weaker, and then so do the reflexes to having its body touched and its skin pricked and pinched. Further, the cat assumes a recumbent position, no longer being in a condition to maintain even the front part of its body in a normal position, and then not the head, either, and, if therapeutic recompression is not undertaken, the animal soon dies. In approximately half of all such cases, soon after the first signs of impaired coordination of movements, paresis and paralyses of the rear extremities and rear part of the body arise, in which case, as is not strange when paresis and paralyses of the rear extremities develop, the animal's general condition often proves to be better and the prognosis for its life more favorable than in cases when a general weakening of the animal sets in at once, without partial paralyses. All of the above-described phenomena, independently of whether or not they are accompanied by paralyses, run their course against the background of hard panting; beginning in a light form soon after decompression, panting in all such cases steadily progresses and attains extreme proportions just when the animal falls into prostration.

The most serious cases of decompression disorders that have just been described, if therapeutic recompression is not undertaken in good time, most often terminate in the death of the animals; more rarely, the cats survive, but are left with persistent flaccid paralyses of the rear part of the body. In lighter cases, the matter is confined to intensive panting, clear depression of the animal and incipient impairment of the

coordination of movements. At this stage, decompression disturbances are completely reversible, and, if we are really dealing with a moderately severe form, they gradually disappear spontaneously, even without recompression. But inasmuch as there is always the danger of subsequent aggravation of functional disorders and inasmuch as the symptoms in question (intensive panting, severe depression of the animal and the signs of uncoordinated movements) are an indubitable indication of pronounced decompression disturbances, they serve as a signal for immediate therapeutic recompression.

In rabbits, the outward picture of incipient decompression disturbances is even poorer than in cats. In the foreground here again are panting and a general inhibition of the animal. In the initial period of the development of decompression disorders, individual rabbits display the above-mentioned symptom of "catching fleas." This symptom seldom appears, but applied to rabbits its significance increases inasmuch as under normal conditions, the practice of "biting out the hair" is not at all characteristic of these animals. In the early period of the subsequent development of decompression disorders, nothing new is added to the above-mentioned signs; all that can be seen is /93 how panting keeps worsening and the rabbit's general depression keeps deepening. Of the latter it is easy to convince oneself by checking the animal's reactions to different immediate stimuli. Blowing into the animal's snout (to which rabbits usually react very energetically), pricking and pinching of the skin or pressing of the tip of the tail do not evoke the usual motor reactions or are even left quite unanswered. Using all of these and other procedures, it is difficult to compel the rabbit to move; panting heavily, with an arrested look, retracting its head into its body, it sits motionless in place, showing complete indifference to its surroundings. Symptoms of the bends type are rarely detected in rabbits and are not clearly expressed: they are betrayed by a functional change in the affected extremity only when the animal moves. Very often, these functional disorders are gradually obliterated and disappear without therapeutic recompression.

Serious decompression disturbances in rabbits occur in two forms. In one, the symptoms break out unexpectedly and suddenly. The rabbit, showing nothing but depression and incremental panting all at once collapses on its side, falls into opisthotonos, then begins to thrash about in convulsions and dies. These clonic spasms are short-lived, but very powerful; they usually terminate in acute opisthotonos, which is also accompanied by squealing; after this, muscular atony sets in: for 1-2 min after the heart has already stopped beating, individual sharp inspirations can be noticed; they become ever rarer and weaker and soon cease entirely. Therapeutic recompression, as experiments show, can help only in the event that it is applied at the very start of these serious symptoms, when the rabbit is just beginning to

weaken and collapse on its side. As a rule, when convulsions have begun, the matter is irremediable, and therapeutic recompression has no effect. It should be noted that this form of decompression disturbances that is very fatal in its suddenness is sometimes preceded by one sign that can put the experimenter on the alert in good time: in the fixedly sitting rabbit there appears the rhythmical retractions of the head that are typical of pronounced altitude hypoxemia. The head is slowly and smoothly raised a little and is retracted more and more until the rabbit's ears are lying on its back; this is followed by a rapid return of the head to its usual position, and everything is repeated from the beginning. When this sign appears, there is every reason to expect the above-described fatal decompression disorders.

The second form of serious decompression disturbances in rabbits is characterized by the development of paralyses of the rear extremities and rear part of the body. Paralyses develop in rabbits considerably more gradually than in cats, and the first signs of them in the form of pareses often already appear in the early period of decompression disorders. In addition /94
to this, it can be noticed that the rabbit in moving gathers its rear extremities under it more sluggishly and slowly and sometimes leaves them stretched out for a while. Then very pronounced paresis sets in, with a noticeable weakening of the muscular tonus, and subsequently, complete flaccid paralysis of the entire rear part of the body. The development of pareses and paralyses in all cases without exception is accompanied by steadily increasing panting. As was established in a study carried out under my guidance by G. A. Lavrova with the special purpose of studying the picture of serious decompression disturbances in rabbits after their stay under a pressure of 4.5 psig and 10 psig, paralyses developed in the overwhelming majority of cases (approximately 2/3); sudden spasms and the death of rabbits, on the other hand, are rarely observed (1/3 of the cases). But very often the paralyses are not confined to the rear part of the body but spread to the front extremities and the neck muscles; in other words, they become general. The development of total paralyses is accompanied by respiratory disorders, followed by respiratory standstill and the animal's death.

According to the data of G. A. Lavrova, these symptoms are followed by the death of approximately half of the rabbits that developed postdecompression paralyses.

In the histologic part of this study carried out by G. A. Lavrova under the guidance of Professor V. P. Kurkovskiy the picture of lesions of the spinal cord was studied in detail in rabbits that had developed spinal paralyses but survived. The results of the histologic investigations bear witness to the aeroembolic (ischemic) nature of postdecompression spinal paralyses.

In mice, the development of decompression disturbances is in general characterized by the same symptoms as in rabbits. The first signs are panting and a loss of the customary mobility of the mice. Further, against a background of increasing panting, either acute general spasms develop that terminate in the animal's death, or pareses and paralyses arise -- for the most part of the entire rear half of the body and, more rarely, of individual extremities. In some animals, decompression disturbances stop at this stage: panting in such mice soon weakens. If the front part of their body is inspected, it looks quite normal, but the rear half of the body turns out to be completely paralyzed: when the mice move, the rear extremities are lifelessly dragged along the surface of the table. Characteristic of decompression disturbances in mice is the rapid dynamics of the functional changes. Whether the decompression disorders progress up to critical disturbances that are incompatible with life, whether paralysis of the rear part of the body develops, or whether the animal recovers -- everything happens and is consumated in a matter of minutes.

It should be noted that in all animals, the nature of the decompression disturbances depends strongly on the magnitude of the overpressure under which the organism has been. In particular, the most serious forms of these disturbances are encountered under a great overpressure.

The fundamental conclusion to be drawn from observations of the outward picture of the development of decompression disturbances in different animals after their stay under increased pressure is that the first functional shifts in all cases without exception involve the respiration and cardiovascular activity and are accompanied by a change in the organism's general condition (more or less pronounced general inhibition and depression). These functional shifts always and invariably precede the development of specific postdecompression symptoms, and in a number of cases are the only result of the decompression effect. The respiratory changes, which at first are barely perceptible, in all those cases where things progress to development of pronounced decompression disturbances increase steadily and change into clear panting. Panting also accompanies all the most serious decompression disturbances, independently of their nature, its intensity generally corresponding directly to the degree of seriousness of the functional disorders. /95

Thus, panting, the changes in cardiovascular activity (judging from dogs) and the concomitant symptoms of general depression of the organism are the earliest signs heralding even more characteristic and constant symptoms of decompression disturbances. These functional shifts are the main backdrop on which the different specific decompression symptoms are superposed in the course of development of caisson phenomena. Getting ahead of myself, I must say that the cause of these functional

shifts resides in aeroembolism of the venous system and pulmonary capillaries. In accordance with A. P. Brestkin's data about the blood's lesser ability, as compared with the tissues, to retain nitrogen in the state of a supersaturated solution, the facts show that after decompression, gas bubbles arise earliest of all in the venous blood and that aeroembolism of the venous system and pulmonary capillaries is the primary link in the pathogenesis of decompression disorders. Panting and the organism's general depression signal the onset of aeroembolic phenomena,[4] and subsequently reflect the dynamics of the latter's development and their general intensity. Direct experimental evidence for this will be produced below. But even independently of this /96 evidence, the very dynamics of the changes in respiration and in the organism's general condition after decompression effects of sufficient intensity leave the clear impression of a continuous ongoing and steadily intensifying process, at certain stages of which, in dogs as well as in human beings, the bends symptoms at first arise with the greatest constancy, while frequent phenomena in the final stages are pareses and paralyses.

All these facts compel critical treatment of the firmly established standard evaluation of the consequences of decompression and of specific decompression symptoms, in particular the bends. When researchers encounter the appearance of these symptoms, they say that the animal fell ill with decompression sickness. What they mean is that as long as there were no specific decompression symptoms, the animal was healthy, but as soon as the symptoms appeared, it became sick. With such an interpretation of the symptoms in question, of course, the first signs of the decompression phenomena that have begun in the organism, signs which often precede these events by much time, escape the influence of researchers and frequently go unnoticed. But if the researcher, before the development of specific symptoms, does encounter some outwardly noticeable functional shifts, in particular general depression and panting, then these shifts are interpreted as no more than a prodrome of the disease and no independent significance is ascribed to them. Actually, however, the facts show that the given functional shifts are a considerably more general, constant and, in this sense, more typical consequence of decompression than the specific decompression symptoms themselves. By no means does it follow from this that the symptoms of the bends must be retained as the criterion for the onset of decompression disorders, or that

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[4] It must be assumed that the initial formation of single fine gas bubbles in the venous blood takes a symptomless course, and that the functional shifts involving respiration and cardiovascular activity, as well as the changes in the organism's general condition, arise only when the aeroembolic phenomena have attained a certain intensity.

one must take one's cue from panting and a change in the organism's general condition. These signs, as has already been said, develop gradually, appear differently in different animals and the degree of their manifestation is difficult to compare in different cases even in one and the same animal, so that they cannot be used as a clear indication of the onset of decompression disturbances. Moreover, as will be shown below, it is in the nature of things that these signs, in particular panting, must not be classified as functional disturbances; they must rather be regarded as defense reactions of the organism, while the symptoms of the bends are obvious functional disorders. Consequently, the symptoms of the bends in those animals in which they develop with the greatest constancy (goats, dogs) can quite legitimately be adopted as the criterion for the onset of decompression disturbances. But these symptoms must not be torn out of the context of all preceding functional shifts and regarded as some independent sickness that suddenly appeared.

It is necessary to be clear about one thing, namely, that the given symptoms reflect only one of the stages of a process /97 that began earlier and is still going on -- the development of decompression phenomena in the organism --, one of the stages of the organism's interaction with an extraordinary factor (gas bubbles), a stage that, together with other physiological shifts, characterizes the onset of manifest functional disorders.

It follows from the above description of the outward picture of the development of decompression disturbances in cats, rabbits and mice that for these animals, it is very difficult to ferret out some absolutely clear-cut criterion for the start of manifest decompression disorders. Decompression phenomena develop in conformity with the natural laws of their dynamics, growing now with more, now with less speed; these dynamics are reflected by the animals' progressive panting and increasing general depression, and subsequently, the onset of uncoordinated movements, paralyses or critical functional disorders of the respiration and blood circulation.

Consequently, in experiments on cats, rabbits and mice, we can speak with complete assurance only of the presence or absence of a given serious and life-threatening decompression disturbance. But as far as the entire preceding period of development of the decompression phenomena is concerned, when they are expressed in terms of more or less severe panting and the animals' general depression, here it proves to be impossible to determine the beginning of the "sickness." The generally accepted alternative formulation of the question -- has "decompression sickness" set in or not -- in these cases confronts the researcher with irresolvable difficulties.

The results of the above-described observations point to the necessity of paying great attention to changes in the breathing,

cardiovascular activity and general condition of human beings after decompression from under increased pressure. It must be assumed that in human beings, the same general principle is valid as in animals, and the beginning of the development of caisson phenomena is accompanied by the same functional shifts. In man, to be sure, owing to his ability to differentiate finely and evaluate accurately his sensations, so early a symptom as itching of the skin acquires the significance of a completely reliable sign of incipient caisson phenomena. It is even possible that this symptom outstrips noticeable changes in breathing and cardiovascular activity. As far as the general condition of the organism is concerned, then, probably everybody who has himself experienced the development of decompression disorders more than once, knows full well the peculiar sensation of general fatigue and depression that precede painful symptoms of the bends type. According to my personal impression, incident to decompression after a stay at a "depth" of 50-60 m executed in accordance with a method sanctioned by the rules of diving work, this sensation sometimes arises after each transition from one halt to another and then gradually weakens and disappears. Obviously, it is no accident /98 that among the first complaints of casualties to appear in descriptions of cases of serious decompression disorders that have been encountered before (cf. in particular, the book of P. Bert), are fatigue, breakdown and general discomfort. And so the study of the outward picture and visible signs of decompression disorders that arise in animals after a stay under increased pressure lead to the following conclusion.

Panting and a general depression of the animals are the most constant signs pointing to the development of decompression phenomena in the organism after a stay under increased pressure. These signs always precede the different specific manifestations of pronounced decompression disturbances. Thus, the appearance of the specific decompression symptoms of the bends in dogs is generally preceded by changes in breathing, heart action and a general inhibition of the animals. In a number of cases, the consequences of decompression are even confined to these functional shifts alone.

CHAPTER 6

PHYSICAL AND PHYSIOLOGICAL CONDITIONS UNDER WHICH DECOMPRESSION DISORDERS ARISE

The experimenter studying the effect of different extraordinary external factors on the organism is generally free to change their intensity and time of action. He holds, as they say, the factors in his hands.

In studying decompression disturbances, the case is somwehat different. Here the experimenter can vary the external conditions on which the development of the decompression disturbances depends, but the factor that is immediately responsible for them -- gas bubbles -- arises invisibly to the naked eye in the organism's internal environment and lends itself neither to arbitrary changes, nor to accurate qualitative determinations. Therefore, in order not to proceed blindly in physiological analysis of decompression disturbances it is necessary at least to know quite accurately the conditions under which free gas bubbles begin to form in the organism's internal environment after decompression. Aimed at giving a more precise definition of these conditions were indeed the two series of experiments presented below. The first of them, aimed at checking the correctness of the idea of the "limit" developed by I. I. Savichev and A. N. Bukharin, was carried out together with A. P. Brestkin and N. Ya. Sidorov.

Checking the correctness of the idea of the "limit" /99

As has already been pointed out (cf. Chapter 3), the sharp polemic that raged around the question of what is the measure of maximum permissible saturation of the organism with an inert gas -- a coefficient or a limit -- could not be regarded as terminated until it had been checked by direct experiments undertaken for that special purpose.

To this end, we conducted the experiments on five dogs and six cats that are presented below. For each animal, we first carried out repeated determinations of those maximal "depths" after a prolonged stay at which and subsequent rapid decompression to the "surface" [5] no manifest decompression disturbances yet arise. The pressure of the nitrogen in the organism (after decompression to normal pressure) that corresponds to these depths is usually called the permissible supersaturation value. This value can be regarded either as a safe supersaturation coefficient or as a "limit." Let us examine, for example, the case where the maximum safe depth for an experimental animal is equal to 14 m. The total pressure corresponding to this depth amounts to 2.4 atm, and the partial nitrogen pressure, to 1.92 atm. The coefficient is the ratio of the pressure of the nitrogen in the organism to normal pressure, i.e., in our case, 1.92/1 = 1.92; the "limit", according to the authors' idea, is expressed in terms of the difference between

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[5] In what follows we will permit ourselves to use the terms "depth" and "surface" without quotation marks inasmuch as in treating questions connected with diving, they are generally accepted, and to translate these expressions into the corresponding pressure units presents no difficulty.

the pressure of the nitrogen in the organism and the overpressure. Inasmuch as the overpressure incident to decompression to the surface comes to zero, the "limit" corresponds to this same value of 1.92. After determining the permissible supersaturation value (PSV) in primary experiments, the animals were placed for 4 or 6 hours at great depths selected so as to be able, without exceeding the found safe supersaturation coefficient (SSC), to transfer the animal to the safe depths established for it, keep it there for another 4 or 6 hours and return it to the surface. Let us clarify this by means of an example based on the figures given above (the maximum safe depth is equal to 14 m, and the corresponding safe supersaturation coefficient is 1.92). We must know from what depth we can transfer the animal without exceeding the coefficient of 1.92 under a pressure of 2.4 atm (depth of 14 m). The coefficient is determined by the ratio of the pressure of the nitrogen in the organism to the total pressure after decompression. Consequently, we must first determine the pressure of the nitrogen (P_x) at the desired level. This is easy to do by the simple calculation $P_x/2.4$ atm = 1.92, whence /100 P_x = 2.4 atm · 1.92 = 4.63 atm. To know the total absolute pressure at the depth of interest to us, we must divide the found nitrogen pressure by 0.8. Undertaking this, we find that it is equal to 4.63 atm/0.8 = 5.57 atm. Correspondingly, the overpressure is equal to 4.75 atm and the necessary depth, to 47.5 m. Transferring the animal from this depth to a depth of 14 m and, after a prolonged stay here, to the surface, we thus get a coefficient of 1.92 in both cases, but if we carry out the calculation according to the limit and subtract its value from the nitrogen pressure at the found depth, we get the manometric pressure, which is equal to 4.56-1.92 = 2.64 psig, i.e., the depth of the first halt amounts to 26.4 m. These divergences between coefficient and limit will be the more considerable, the greater the animal's permissible supersaturation value and the maximum depth at which it found itself in experiments involving two-step decompression.

The results of experiments on dogs are shown in Table 1. In Column 2 are shown the depths corresponding to the animals' PSV.[6] In Column 3 are shown the pressure of the nitrogen in the organism that corresponds to these depths, which, as has already been pointed out, can be regarded either as a coefficient or as a limit. In Column 4 are shown the maximum depths at which the animals were kept, in Column 6, the values of the SSC used for

[6] The accuracy of determination of these depths amounted to 2 m H_2O, or 0.2 atm. Recorded in some dogs in the course of the experiments was an increase in the given depth that was connected with the process of conditioning the organism to decompression effects, which had not been terminated. The question of conditioning will be examined separately in one of the following chapters.

TABLE 1. RESULTS OF EXPERIMENTS ON DOGS LOWERED TO GREAT DEPTHS

Date of experiment, 1959	Depth corresponding to the animal's PSV,* m	Coefficient or limit	Depth of immersion, m	Exposure, h	SSC** used in experiment	Depth of actual halt according to SSC, m	Value of limit incident to transition to first halt	Depth of first halt according to the established limit, m	Symptoms of decompression disorders	
									at the first halt	at the surface
					Mishka					
2.XI	12	1.76	57	5—4	1.92	18	3.60	36.4	Some	—
9.XI	12	1.76	38	0—4	1.74	12	2.64	20.8	None	None
16.XI	12	1.76	57	5—4	1.74	21	3.30	36.4	"	Gradual decompression
23.XI	14	1.92	47	0—4	1.90	14	3.16	26.4	"	Some
17.XII	16	2.08	57	5—4	2.08	16	3.80	33.2	"	"
					Sedov					
13.XI	12	1.76	38	0—4	1.74	12	2.64	20.8	"	None
26.XI	14	1.92	47	0—4	1.9	14	3.16	24.6	"	"
2.XII	16	2.08	57	0—4	2.06	16	3.76	32.8	"	"
					Ryzhik					
18.XI	16	2.08	57	5—4	2.08	16	3.80	33.2	"	Some
24.XI	16	2.08	38	0—4	1.74	12	2.64	17.6	"	None
					Bogatyr					
19.XI	18	2.24	68	0—4	2.22	16	4.64	40.0	Some	—
27.XI	18	2.24	47	0—4	1.9	14	3.16	23.2	None	None
8.XII	18	2.24	57	5—4	2.08	16	3.80	31.6	"	"
					Rex					
1.XII	20	2.40	57	5—4	2.08	16	3.80	30.0	"	Some

*PSV -- is the permissible supersaturation value. **SSC -- is the safe supersaturation coefficient.

TABLE 2. RESULTS OF EXPERIMENTS ON SIX CATS LOWERED TO GREAT DEPTHS

Date of experiment, 1959	Depth corresponding to the animal's PSV, 7 m	Coefficient or limit	Depth of immersion, m	Exposure, h	SSC[8] used in experiment	Depth of actual halt acc. to SSC, m	Value of limit incident to transition to first halt	Depth of first halt according to established limit, m	Symptoms of decompression disorders	
									at the first halt	at the surface
					№ 1					
1.VI	22	2,56	90	0—4	2,50	22	5,8	54,4	Death from decompression disorders	
					№ 2					
21.VII	28	3,04	90	0—6	2,35	24	5,6	49,6	None	None
2.X	28	3,04	90	0—6	2,5	22	5,3	49,6	"	"
1.XI	28	3,04	85	0—6	2,37	22	5,4	45,6	"	"
11.XI	28	3,04	105	4	2,7	24	6,8	61,6	"	"
19.XI	28	3,04	120	4	2,89	26	7,8	73,6	"	Some
					№ 3					
5.XI	28	3,04	90	6	2,2	26	5,4	49,6	Some	—
					№ 4					
28.IX	24	2,72	75	6	2,27	20	4,8	40,8	None	None
9.X	24	2,72	75	5	2,27	20	4,8	40,8	"	"
10.IX	24	2,72	92	4	2,56	22	6,0	54,8	Some	—
20.XI	24	2,72	92	4	2,56	22	6,0	54,8	None	None
					№ 5					
11.XI	22	2,56	75	4	2,8	14	5,4	42,4	Death from decompression disorders	—
					№ 6					
9.XI	26	2,88	75	4	2,27	20	4,8	39,2	None	None
16.X	26	2,88	92	5—4	2,56	22	6,0	53,2	"	"
26.XI	26	2,88	92	5—4	2,56	22	6,0	53,2	"	"

the calculation, in Column 7, the depth of the first actual halt. Column 8 reflects the difference between the pressure of the nitrogen in the organism at the depth and the manometric pressure at the first halt, i.e., the limit as calculated according to the principle proposed by I. I. Savichev and A. M. Bukharin. In Column 9 is shown the depth of the first halt, calculated according to the limit. The last two columns show the results of observations of the development of decompression disturbances in the animals. As was pointed out earlier, regarded as incontestible signs of decompression disorders in dogs are the symptoms of the bends; in cats, these same symptoms, or an abrupt change in their general condition, which is manifested in uncoordinated movements, pronounced general inhibition and intensive panting.

The experimental results of Table 1 show that at the first halt, decompression disturbances arose only in three experiments out of 14, the coefficient used in the calculation being higher than the safe supersaturation coefficient in one case (experiment 2.XI). At the same time, the actual depth of the first halts were considerably less than the depths calculated according to the limit. After returning to the surface, decompression disturbances arose in four cases out of 11, while the remaining seven experiments took their course without these disturbances from beginning to end.

The results of analogous experiments on cats are shown in Table 2. In this series of experiments, as a rule, we used values of the safe supersaturation coefficient that are less than those established in the preliminary investigations.[7] Two experiments constituted an exception: in one (experiment 1.VI), the coefficient was not decreased, while in the other (experiment 11.XI), it was increased. These experiments both terminated in the animal's death at the first halt. Out of the remaining 13 experiments, decompression disturbances arose at the first halt only in two cases. Here the actual depth of the first halt diverged from those calculated according to the limit even more than in the experiments on dogs, in spite of the fact that the value of the limit used in the calculations was not decreased. In only one of the 11 experiments that were carried through to the end did decompression disturbances develop in the animal after final decompression. In the remaining ten experiments, both decompression steps elapsed without signs of decompression disorders.

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[7] This was done because, according to the available data [Baycott, Damant and Haldane, 1908; Brestkin, 1952; Zal'tsman and Zinov'yeva, 1964] and the practice of calculating decompression tables, the safe supersaturation coefficient decreases with increasing pressure of the dissolved indifferent gas.

These experimental results confirmed the fact that the real measure of the supersaturation of the organism's fluids and tissues with an inert gas is the coefficient, i.e., the ratio of the gas pressure to the surrounding pressure, and not the difference between these values. They compel rejection of the idea of the limit as the main condition for the onset of decompression disturbances. Indeed, in transferring the animals from great depths to the first halt, we exceeded the value of the limit by a factor of 1.5-2 and even more (compare the figures shown in Columns 3 and 7 of the table) and in the great majority of cases, no decompression disorders were recorded.

Consequently, there can be no question of the limit being constant and independent of depth.

These data show at a glance that the use of a constant value of the limit incident to decompression from under great pressures entails an increase in the first halts, as a result of which decompression time must be substantially increased. Thus, for example, if we had been guided by the constancy of the limit in raising the cat of experiment 11.XI from a depth of 105 m, then not one , but two four-hour halts would have had to be made: the first at a depth of 61.6 and the second, at 28 m. All of this points to the fact that the idea of the limit is false, and that in calculating decompression methods we must be guided by the coefficient. Moreover, in conformity with the above-mentioned data, the experiments show that the safe supersaturation coefficient cannot be regarded as being invariable for different conditions: at great depths, it decreases somewhat. For the /104
time being, the question of whether this change in the coefficient is mainly based on physicochemical principles or on physiological circumstances connected with the effect of depth factors on the organism, remains open.

Determination of the physical coefficient of maximum permissible supersaturation of the orgamism with nitrogen for different animals

In the just-described experiments, the degree of safe and maximum permissible supersaturation of the organism with nitrogen was determined according to physiological criteria, namely, the absence or appearance of pronounced symptoms of decompression disorders. In conformity with this, the true measure of safe and maximum permissible supersaturation established in the experiments can be classified as the physiological coefficient of safe and maximum permissible supersaturation of the organism with nitrogen.

It is well-known that animals of different species differ very sharply in their resistance to decompression disturbances. Thus, the resistance of dogs and rabbits exceeds that of man

comparatively little, the resistance of cats turns out to be considerably higher, and that of rats and mice, still much higher. These small animals easily endure decompression conditions that in terms of depth, exposure times and time and magnitude of drop in pressure exceed by far those that are fatal for man. Thus, it can be said that the physiological coefficient of safe or maximum permissible supersaturation differs sharply for different animals. In this connection, it was natural to set oneself the task of determining for different animals those maximum permissible degrees of supersaturation of the organism with nitrogen at which the process of gas-bubble formation in the blood begins. We have in mind gas bubbles that are discernible to the naked eye, i.e., bubbles of such a size as to acquire pathogenic significance according to all the data. In contrast to the physiological coefficient of safe or maximum permissible supersaturation, here we can speak of the physical coefficient, i.e., that limit of supersaturation at which the process of manifest gas formation begins. In considering the causes of the sharp differences in resistance of different animal species to decompression disturbances, the question may arise whether these differences are not connected with different abilities of the animals' tissues and blood to retain an inert gas in the state of a supersaturated solution. Independently of this, it is of interest as applied to all animals to elucidate what are the relations between the physiological and physical coefficients of maximum permissible supersaturation. In this connection, the experiments presented below were undertaken.

TABLE 3. COEFFICIENT OF MAXIMUM PERMISSIBLE SUPERSATURATION OF THE BLOOD FOR DIFFERENT ANIMALS /105

Pressure, psig	Exposure time, h	Cats	Rabbits	Guinea pigs	White rats	White mice	Pigeons	Gas bubbles in the blood
2.5	4-5	1	1	1	4	2	-	Great amount in all cases
2.2	4-6	-	1	1	4	1	1	Considerable amount in all cases
2.0	4-6	1	2	-	7	2	-	Moderate amount in all cases
1.8	6	-	1	-	1	1	-	Comparatively small amount in all cases
1.6	7	-	1	-	1	2	1	Single bubbles in all cases
1.4	7	-	1	-	1	2	-	Single bubbles in three cases
1.2	7-9	-	1	-	2	2	1	None detected in any case

Used in the experiments were cats, rabbits, guinea pigs, white rats, white mice and pigeons -- a total of 47 animals. The animals were placed in a recompression chamber in different experiments under different pressures (1.2-1.4-1.6-1.8-2.0-2.2-2.5 psig) and kept there from four to nine hours. Fifteen-twenty minutes before the end of this period, the experimenter entered the chamber through the airlock and killed the animals in it by administering fatal doses of hexenal or by pricking their medulla oblongata with a needle. After this, the corpses of all the animals were subjected to rapid decompression to the surface, where autopsies were performed on them and careful observations were conducted of different regions of the body (mainly the different vessels) in order to detect gas bubbles in them. Excluded from the experiments in this manner were any physiological factors whatsoever that might affect postdecompression gas formation, and on the basis of the appearance of gas bubbles, the coefficient of maximum permissible supersaturation was determined for the blood and tissues of different animals under the condition of identical and complete saturation of their organism with nitrogen. The results of the experiments are shown in Table 3.

In these experiments, as can be seen from the table, no gas bubbles were detected in the blood of the animals' corpses at a pressure of 1.2 psig. The appearance of single bubbles was recorded in a rabbit, a rat and a mouse at a pressure of 1.4 psig; at a pressure of 1.6 psig and higher, bubbles appeared in the blood of all animals, their number increasing regularly with increasing pressure. The times for appearance of bubbles in the blood varied from 10-15 min to an hour and a half and were the shorter, the higher the pressure used in the experiment. In experiments of this kind, no place in the vascular system where bubbles arose preferentially (oftener and sooner) could be noted. Gas formation was noted in different cases now in some, now in other vessels, /106 both venous and arterial. After pressures of 2.0-2.2 psig, the appearance of gas bubbles in the serous fluids becomes marked (they probably arise here no later than in the blood, but the conditions for discerning them with the naked eye in transparent media are worse than in the blood), and after high pressures, in other tissues, too.

These experiments bear witness, as it were, to the fact that the physical supersaturation coefficient for the blood of different animals corresponds to a depth of 12 m (a pressure of 1.2 psig). In view of the insufficient number of experiments, however, it is quite impossible to speak of this categorically. An indubitable conclusion that can be drawn from these experiments, in spite of their limited number, is that there are no significant differences in the maximum permissible degrees of supersaturation at which the process of visible gas formation develops in the blood of animals of different species. Indeed, beginning with a pressure of

1.6 psig and higher, bubbles that are visible to the naked eye appear in the blood of all of the animals, and their number also increases with increasing pressure approximately to the same extent in all of the animals. Hence it follows that it is quite impossible to connect the extremely great differences in the resistance of animals of different species to decompression disorders with any sharp differences in the ability of their blood (as well as their tissues) to retain nitrogen in the state of a supersaturated solution. Generally speaking, this conclusion should already have been obvious. But this does not obviate the necessity of its experimental confirmation.

Returning to the question of the limit of maximum permissible supersaturation as a physical magnitude, it is necessary to adduce data recently obtained in our laboratory by R. T. Kazakova. In this same type of experiment conducted on rats, she made an attempt to determine this limit more accurately. The rats were kept under increased pressure for six hours, and then killed in the chamber; their rapid single-step decompression to the surface was effected and here, after section of the corpses, for two hours a careful watch was kept for the formation of gas bubbles in the blood. The following results were obtained:

Pressure	Number of animals	Presence of bubbles	Pressure	Number of animals	Presence of bubbles
1.2 psig	10	9	0.8	20	5
1.0	16	3	0.6	47	1

It can be seen from the above data that, firstly, no absolutely clear-cut value was obtained for maximum permissible supersaturation and, secondly, under the conditions of these experiments, the limit of maximum permissible supersaturation dropped toward lower pressures and correspondingly lesser degrees /107 of supersaturation. Recorded was the fact of appearance of gas bubbles after a stay under a pressure of no more than 0.6 psig. True enough, this was noted only in one case out of 47, the net result being the detection of no more than a few fine bubbles. At pressures of 0.8-1.0 psig, again no more than single gas bubbles were found in 1/4-1/5 of the cases; at a pressure of 1.2 psig, they were already detected in somewhat greater, but by no means mass, numbers in almost all cases. Hence, it follows that, as applied to these experimental conditions, it is impossible to speak of the physical coefficient as of a strictly determinate value. Similar determinations of the limit of maximum permissible supersaturation in a rarefied atmosphere, which were undertaken by K. S. Yurova and R. T. Kazakova and which will be discussed later, created the impression of considerable individual differences in the process of gas formation in animal blood under these experimental conditions.

An incontestible inference to be drawn from these and analogous experiments with a rarefied atmosphere is that bubbles that are visible to the naked eye appear in the blood only when the degree of supersaturation of the organism as a whole and of the blood exceeds certain limits. The minimum permissible supersaturation at which, in a few individual cases, single gas bubbles appear in the blood prove to be less than those degrees of supersaturation at which the formation of rather considerable numbers of gas bubbles already becomes the general rule. Obviously, it is these latter values of supersaturation that, practically speaking, ought to be adopted as the physical coefficient of maximum permissible supersaturation. Especially important from the practical, as well as theoretical, point of view is the circumstance that when the blood is supersaturated with less than these minimum values, gas bubbles that are visible to the naked eye never arise.

Thus, the most important conclusion to be drawn from all these experiments is that there exists an increased pressure range that is safe from the point of view of formation of more or less large gas bubbles (i.e., bubbles that are visible to the naked eye) in the blood after decompression; this is followed by another pressure range, in which the postdecompression formation of such bubbles is possible in individual cases and, with increasing pressure, becomes ever more probable; and, finally, we have a rather clear-cut value of pressures at which pronounced gas formation in the blood becomes a general rule extending to animals of different species. The value of these pressures is, on the average, in the range from 1.4-1.6 psig.

It is of particular interest to compare the supersaturation coefficient as a physical value with the supersaturation coefficient as a physiological value -- i.e., one that determines decompression disorders. /108

Thus, it turns out that the physiological coefficient of safe supersaturation is just about as much above the physical coefficient in people as it is in rabbits and dogs.

This discontinuity between the safe supersaturation coefficient and the physical coefficient in cats proves to be even greater, while in small animals (rats, mice), it is so great that the corresponding values are generally incomparable. These animals show no decompression symptoms even after a stay at depths of 40-50 m.

In explaining all of the facts enumerated above, it is necessary to take into account the following two circumstances.

The first circumstance that suggests itself to explain the regular discontinuity between the physical and physiological

coefficients of safe supersaturation is the symptomless course of incipient aeroembolic phenomena. If the physical coefficient of supersaturation of the blood is barely passed the formation of gas bubbles in it is inevitable; but physiological reactions and functional disorders will arise only when the aeroembolic phenomena attain a certain intensity. This may explain the considerable, but not excessive, divergences between the safe supersaturation coefficient and the physical coefficient that can be observed in human beings, as well as in dogs and rabbits.

The second circumstance, which concerns mainly small animals, has to do with the rate of desaturation of the organism from inert gases, especially nitrogen. There can be no doubt that immediately after rapid decompression from considerable depths, the venous blood, and in a number of cases the tissues, too, turn out to be supersaturated with nitrogen above the physical coefficient. But it takes some time for gas bubbles of considerable size to be formed, and during this time, incident to very rapid desaturation of the organism from nitrogen, the supersaturation of the blood and tissues may drop below dangerous levels.

Here a role is played, on the one hand, by the absolute quantity of excess nitrogen dissolved in the body and, on the other, the rapidity with which this nitrogen is washed out of the tissues and given off to the external environment. In small animals, owing to their limited volume, the absolute quantity of nitrogen is not very great, and the rate of its removal, owing to the intensity of the blood circulation and respiration, is considerable. This may also create the conditions for the excessive supersaturation that arose after rapid decompression to drop down to safe levels sooner than pronounced aeroembolic phenomena can manage to develop.

Be that as it may, it indubitably follows from the foregoing data that for the physical coefficient of supersaturation to be exceeded does not yet mean the inevitable development of decompression disorders, that the coefficient of supersaturation that /109 is safe from the standpoint of decompression symptoms is always higher than the physical coefficient and that this gap is due to physiological causes. The latter has so far been studied scarcely at all and urgently requires experimental analysis and elucidation.

The factual material presented in this chapter permits the following conclusions to be drawn.

The idea of the limit is incorrect. The true measure of the maximum permissible supersaturation of the organism with an inert gas is a coefficient, i.e., the ratio of the pressure of the inert gas in the organism to the surrounding pressure.

With increasing pressure of the dissolved inert gas, the value of the safe supersaturation coefficient decreases, which is

in conformity with the data of other authors.

The coefficient of maximum permissible supersaturation with nitrogen as a physical value is practically the same for animals of different species. The striking differences in their resistances to decompression disturbances depend on physiological properties of the organism, and, in the first place, the circulation of the blood and the respiration.

The coefficient of safe supersaturation of the organism with nitrogen in all animals, as well as in man, to a greater or lesser extent exceeds the physical coefficient of maximum permissible supersaturation that determines the start of formation of gas bubbles that are visible to the naked eye.

CHAPTER 7

CHANGE IN THE ORGANISM'S RESISTANCE TO DECOMPRESSION DISTURBANCES INCIDENT TO REPEATED DECOMPRESSION EFFECTS

The cardinal and most important question facing physiological analysis of decompression disturbances is whether the organism possesses adaptive resources for such an extraordinary factor as gas bubbles in its internal environment, i.e., whether it is able to enhance its resistance to decompression disturbances incident to repeated decompression effects. If this is so, then together with the solution of this question, the task of subsequent investigations will be clear: to elucidate the physiological reactions on which the organism's adaptation to decompression gas formation is based, and to determine the conditions that are most conducive to a perfect course of these reactions.

The question of the organism's ability to adapt to the /110
harmful effect of gas bubbles is of profound theoretical interest inasmuch as it is a question of a factor that is completely new to the organism, one that has never been encountered in the course of evolution. At the same time, it has also a direct practical significance in connection with the problem of conditioning divers and regulating their underwater work.

Finally, the solution of this same question is also very important for those experimental studies which investigate how the development of decompression disturbances depends on different additional effects on the organism. Inasmuch as in this case, repeated experiments with decompression on the same animals are inevitable, it is absolutely necessary to know how these repeated decompression effects, taken in themselves, affect the organism's resistance to decompression disturbances.

In spite of all its importance, the question of the organism's active physiological defense against the effect of gas bubbles that are being formed in it and its adaptation to repeated decompression effects, has not only not been treated experimentally, but has not even been posed by researchers in any clear-cut manner.[8] As has already been noted in connection with our survey of the literature, resistance to decompression disturbances (or, as is more often said, sensitivity and susceptibility to decompression sickness) is connected first and foremost and, in a number of cases, even exclusively, with the ability of the tissues and blood to retain the excess nitrogen dissolved in them. But inasmuch as it is difficult to study closely this ability of the tissues and the factors that affect it and authors do not set themselves such goals, the thinking of researchers has not been stimulated to look for other explanations. If, in discussing the organism's resistance to decompression disturbances and its changes, the question of physiological reactions comes up at all, then it is almost exclusively in connection with saturation and desaturation processes, and not at all from the standpoint of the fight that the organism may be waging with caisson phenomena that are already developing in it. But as far as the functional shifts that take place in the organism incident to the development of caisson phenomena are concerned, all of them, and especially functional shifts involving breathing and cardiovascular activity, are regarded only as direct manifestations of decompression "sickness," i.e., as purely pathologic symptoms. It would seem that if the position that decompression disturbances are a true disease can even be maintained, one ought to ask oneself which of its manifestations are really pathologic, and which of them have a compensatory significance. But, in evaluating decompression disorders, the /111
hypothesis that in the case of every disease, it is necessary to distinguish between pathologic functional disturbances and defense reactions which was postulated in its day by the founders of domestic pathology and physiology (i.e., Pavlov, V. V. Pashutin, P. M. Al'bitskiy) and is nowadays generally known, for some reason or other proves to be quite forgotten.

The study of the organism's adaptive defense resources incident to decompression disturbances was the main purpose of our research.

To achieve this purpose it was first of all necessary to elucidate whether the organism's resistance to decompression disturbances can be enhanced, if it is subjected to repeated decompression effects of gradually increasing intensity. To this very

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[8] We noted the presence in the organism of exceptionally great defensive forces in regard to the noxious effect of decompression aeroembolism in 1958 in a joint article with I. I. Vavilov (Functions of the organism in a changed gaseous medium).

question are addressed the experiments presented below, which were carried out in a number of versions.

Experiments with repeated determination of the safe supersaturation coefficient

Many researchers have undertaken to determine the safe supersaturation coefficient in different animals (in the literature it is usually called the permissible supersaturation value). In so doing, they all used one and the same order of determinations: increasing the pressure from experiment to experiment (usually by 0.2 atm), the authors went as far as that value of the pressure at which caisson symptoms developed in the animal after decompression, then reversed the experiment to the previous pressure (by 0.2 atm), satisfied themselves that the symptoms were absent and thereupon terminated the determination, considering that the limit of the organism's ability to resist decompression disturbances had been found. They did not ask themselves whether the safe supersaturation coefficient would change when its determinations were repeated in succession. But this is precisely the question we asked ourselves.

The initial experiments on three dogs (Zor'ka, Sokol and Valet) were carried out in collaboration with V. A. Aver'yanov and A. A. Savich. The animals were kept under pressure for six hours, after which decompression down to normal pressure was effected as rapidly as possible. It took 60-100 sec. The pressure in the first experiment amounted to 1.2 psig (a depth of 12 m); in every subsequent experiment, it was increased by 0.2 atm, right up to the appearance in the dogs of clear decompression disturbances (symptoms of the bends). Then the experiment was repeated with this same pressure, after which a pressure lower by 0.2 atm was used, i.e., a retrogression by one step down was effected, and determinations were continued in the same order. As soon as decompression disturbances developed, so-called therapeutic recompression was used, as a result of which symptoms of /112 the bends disappeared leaving no trace in any of the cases without exception.

The results of the experiments are shown in Fig. 19. Individual experiments are represented in this figure by columns whose heights show the value of the pressure used. The white columns correspond to experiments that terminated without decompression disturbances and the hatched columns, to experiments in which these disturbances did occur.

As the figure shows, initially decompression disturbances arose in Zor'ka after a pressure of 1.8, in Sokol, 2.2 and Valet, 2.4 psig. A repetition of this experiment with the same pressure values was also accompanied by the development of decompression disturbances; in the following experiments, however, where the

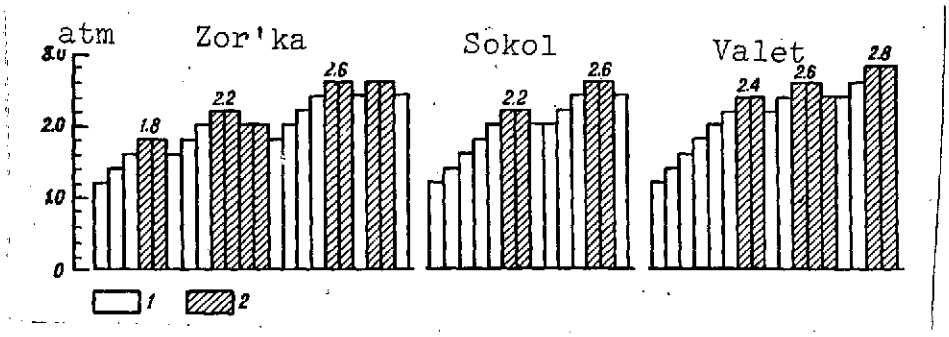

Fig. 19. Change in resistance of dogs to decompression incident to repeated determinations of the permissible supersaturation coefficient

1 -- without disturbances; 2 -- with disturbances.

pressure was reduced by 0.2 atm, decompression disturbances were absent. Had we adhered to the above-mentioned usual order of determinations, we would have had to confine ourselves to these, considering the maximum permissible safe pressure to be 1.6 for Zor'ka, 2.0 for Sokol, and 2.2 psig for Valet (corresponding to safe supersaturation coefficients of 2.08, 2.4 and 2.56). But, as can be seen from the adduced data, in the course of subsequent experiments, the resistance of all three dogs increased regularly and considerably: Zor'ka and Valet painlessly endured experiments with pressures of 2.6, and Sokol, 2.4 psig. Subsequently, all three dogs were used in experiments with the established maximum permissible pressure values and with different additional effects (administration of drugs before decompression, running on a treadmill afterward). These experiments alternated with control experiments in which the safe supersaturation coefficient was checked.

Experiments showed that in all three dogs, the resistance to decompression disturbances became fixed at the attained level, while in Zor'ka, in one of the control experiments, it proved to be increased by yet another step (no decompression symptoms appeared after a pressure of 2.8 psig). The maximum permissible /113
safe pressures in the course of the experiments increased for Sokol and Valet by 0.4 psig, and for Zor'ka by an entire atmosphere;

the safe supersaturation coefficient increased correspondinly for Sokol from 2.4 to 2.72; for Valet, from 2.56 to 2.8 and for Zor'ka, from 2.08 to 2.88 (in one experiment, 3.04).

Thus, it was established in these experiments that, provided we begin determining the safe supersaturation coefficient with pressures that are known to be safe, come down to lower pressures after those experiments in which there were caisson symptoms, gradually increase them again, and continue the experiments, then a considerable enhancement of the dogs' resistance to decompression disturbances takes place.

The facts show that there are practically no exceptions to this principle. It appeared in all of the experimental dogs of V. A. Aver'yanov, who carried out experiments at the same time we did.

In a study by K. S. Yurova, which was conducted under our guidance, the author determined the safe supersaturation coefficient in nine dogs, adhering to the same principle in setting up further experiments. An increase in resistance was noted in all the animals without exception. It corresponded, on the average, to an increase in the safe pressure by 0.4-0.6 atm. In subsequent experiments by K. S. Yurova on five new dogs, the safe supersaturation coefficient was determined according to the same principle, and all the animals showed an increase in it corresponding to an increase in the total pressure by 0.2-0.8 atm.

Thus, the foregoing results of our experiments receive ever newer confirmation , and so far, no case has been encountered which might contradict them.

In five dogs, we alternated the experiments involving determination of the safe supersaturation coefficient with "descents" to great depths (we have in mind experiments using high pressures), which are described in the preceding chapter.

The results of these experiments are shown in Fig. 20. As the adduced data shows, even in this case three dogs showed an increase in the safe supersaturation coefficient, i.e., an increase in resistance to decompression disturbances, while in the other two it did not change. Thus, the result here proved to be inconstant, which is probably connected with the greater intensity of decompression effects incident to ascents from considerable depths and, perhaps, also with the fact that these effects are complicated by the toxic action of compressed nitrogen.

As was already mentioned earlier, all the above eight animals, and 12 other dogs that were used in our research, served in experiments involving decompression effects for very long times (from two to six years). In the investigations, they were

subjected to decompression effects of different kinds and intensities, but in all of them, the value of the safe supersatura-/114
tion coefficient was determined and checked more or less often. These determinations showed that during those working periods when the dogs were subjected to systematic decompression effects of not excessive intensities, the safe supersaturation coefficient in all of them decidedly kept -- with some fluctuations -- to the increased level and appreciably exceeded the values it had in the given dog incident to the first determination.

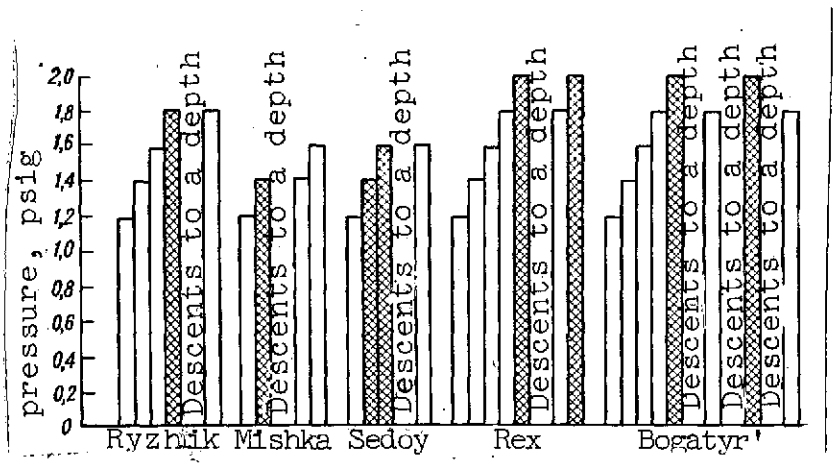

Fig. 20. Change in the safe supersaturation coefficient in dogs after descents to great depths

1 -- without decompression disturbances; 2 -- with disturbances

Table 4 shows the values of the safe supersaturation coefficient obtained for all 20 investigated dogs incident to initial determinations (first cases of decompression disorders) and during systematically repeated decompression effects. As can be seen from the table, the data are quite monotypic. Their statistical evaluation bears witness to the fact that the inference of an increase in the safe supersaturation coefficient under the action of systematically repeated decompression effects is absolutely correct.

The usual decompression disturbances (symptoms of the bends) that arose periodically in the animals did not prevent the safe supersaturation coefficient from keeping to the

increased level in the course of systematic experiments provided they were liquidated in good time by means of recompression.

In those cases where the animal experienced such disturbances many times running, or where, in one or another of the experiments, they proved to be particularly serious, the safe supersaturation coefficient decreased, though, as a rule, it did not drop below its initial value. After a long interruption in the experiments with decompression effects (longer than one or one and one half months), the safe supersaturation coefficient returned to its initial low values. Finally, here is the last point: incident to periodic determinations of the coefficient, it was observed in a number of cases to undergo fluctuations corresponding most often to no more than 0.2 atm of the total pressure, which were not connected in any obvious way with decompression effects themselves, but were apparently determined by changes in the physiological condition of the animals under the effect of some other factors.

TABLE 4. INCREASE IN RESISTANCE OF DOGS TO DECOMPRESSION DISTURBANCES INCIDENT TO SYSTEMATICALLY REPEATED DECOMPRESSION EFFECTS /115

Dog	Maximum safe depth, m		Safe supersaturation coefficient	
	incident to initial determination	subsequently	incident to initial determination	subsequently
Zor'ka	16	28	2.08	3.04
Sokol	20	26	2.4	2.88
Valet	22	24	2.56	2.72
Mishka	14	22	1.96	2.56
Ryzhik	16	24	2.08	2.72
Bogatyr'	18	22	2.24	2.56
Sedoy	14	18	1.96	2.24
Druzhok	20	26	2.4	2.88
Belka	18	24	2.24	2.72
Volchok	22	28	2.56	3.04
Kudryash	20	24	2.4	2.72
Timoshka	18	24	2.24	2.72
Manyunya	16	22	2.08	2.56
Yashka	22	28	2.56	3.04
Snezhok	18	26	2.24	2.88
Donka	18	26	2.24	2.88
Nayda	16	24	2.08	2.72
Chernysh	20	28	2.4	3.04
Mean	18	26	2.24	2.88
Ryzhka	20	26	2.4	2.88
Srednive	18.3	24.8	2.26	2.73
Fluctuations	14—22	18—28	1.96—2.56	2.24—3.04

Change in safe staying times of animals at medium and great depths incident to repetition of experiments /116

The second version of the experiments aimed at studying the organism's resistance to decompression effects incident to their systematic repetition was experiments with single-step decompression of the animals from medium and great depths. In these experiments, the criterion for the organism's resistance to decompression disturbances was the longest time the animal could stay at the depth without decompression disturbances arising in it after rapid decompression to the surface. The longer this time, the more complete the organism's saturation with nitrogen, which means the greater the degree of supersaturation of the blood and tissues after decompression. Consequently, the longer the safe staying time at the depths, the higher the organism's resistance to decompression disturbances.

Systematic experiments with a pressure of 4.5 psig (a depth of 45 m) were carried out on three dogs (Volchok, Kudryash and Timoshka). In the first experiments, the staying time of the animals at the depth was known to be safe (10-15 min); in every subsequent experiment, it was increased by 5 min, right up to the onset of decompression disturbances. After the experiment accompanied by the development of decompression symptoms, we used an exposure that was shorter by 5 min and then gradually increased it again. In a word, we adhered to the same principle as in the case of the determination of the safe supersaturation coefficient: gradual increase in decompression effects, their temporary weakening after the experiments involving decompression disturbances and subsequent gradual increase. For each dog, the experiments were separated by intervals ranging from three to five days. In the event that decompression disturbances developed, we used, as always, recompression. It ensured the complete liquidation of decompression symptoms in all cases with the exception of one (cf. below).

As can be seen from Fig. 21, decompression disturbances arose in Volchok for the first time after a 45-minute stay under pressure. When, however, after reducing the exposure, it was again gradually increased in the next experiment, decompression disorders appeared only after a 65-minute stay of the dog at the depth, i.e., the animal's resistance to decompression disturbances increased sharply. In the following experiment with a 60-minute exposure, decompression disorders of a particular and unfavorable kind also developed. Symptoms of paresis of the rear part of the body suddenly appeared in the dog and began to progress very rapidly. Recompression, which did not have a complete therapeutic effect the first time, was repeated, but again did not completely liquidate the disturbances: a very pronounced paresis of the rear right extremity persisted in this dog. The dog was not

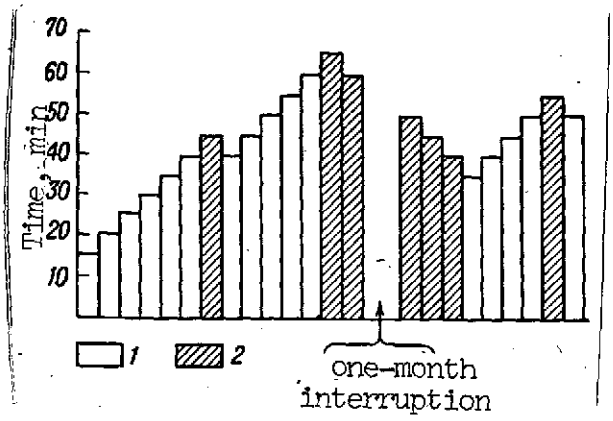

Fig. 21. Change in resistance to decompression incident to repeated effects in Volchok. Value of overpressure in all cases: 4.5 psig; decompression: single-step, at a rate of 1 atm every 20 sec.

1 -- without decompression disturbances; 2 -- with disturbances.

subjected to experiments for a month, and the manifestations of paresis gradually smoothened out and disappeared practically completely. Experiments conducted after this (cf. Fig. 21) showed that the animal's resistance to decompression disturbances decreased sharply and turned out to be lower than it was originally; subsequently it increased again and exceeded its initial value. /119

In our initial experiments with Kudryash (Fig. 22), we diverged from our adopted rule: in spite of the development of decompression disturbances, the exposure in the next experiments was not decreased. Obviously, as a result of this, the animal's resistance to decompression disturbances for a long time did not increase: subsequently, when we began to adhere strictly to the adopted order of applying decompression effects, this dog's resistance to them also increased /118

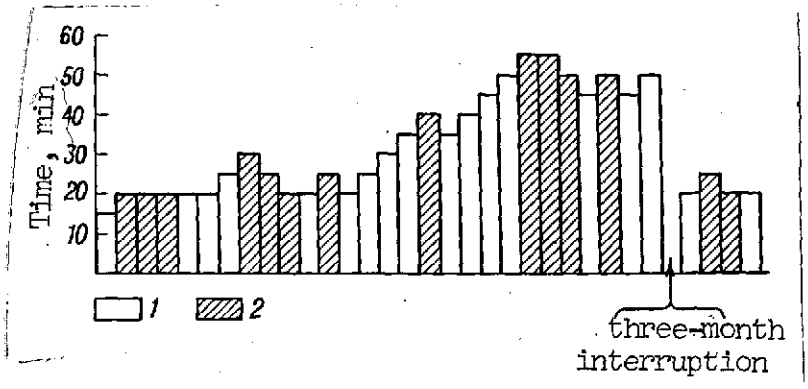

Fig. 22. Change in resistance to decompression incident to repeated effects in Kudryash. Experimental conditions and notation the same as in Fig. 21.

considerably and, as long as the experiments proceeded systematically, kept to its new, high level. After a three-month interruption in the experiments, however, the dog's resistance to decompression disturbances dropped to its original values.

Of interest are the results of subsequent experiments on this dog (Fig. 23). These experiments were supposed to answer the question: can an animal's resistance to decompression disturbances be enhanced by repeatedly applying the same subliminal decompression effects without increasing their intensity from time to time, as was done in the preceding experiments?

The experiments in question were set up every day; in their course, as can be seen from Fig. 23, initially the exposure was increased from 20-25 min, and then, after the onset of decompression disorders, we twice changed over to shorter (20-, and then 15-minute) exposures.

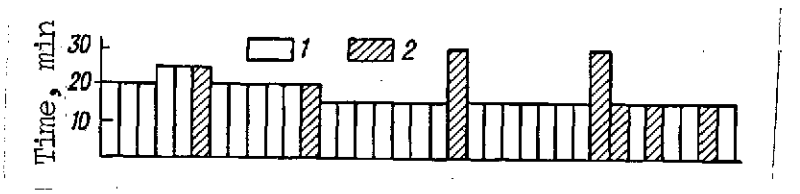

Fig. 23. Change in resistance to decompression incident to repeated effects in Kudryash. Experimental conditions and notation the same as in Fig. 21.

Follow-up experiments with a 30-minute exposure of the dog to pressure, which were set up after 19 and 27 daily effects, showed no increase in resistance for a given exposure -- in both cases, the dog developed pronounced decompression disturbances.

To obtain the effect, a greater number of daily subliminal decompression effects may be necessary. This idea is suggested by the results of analogous experiments on a third dog, to which we will turn presently. But, in any event, there can be no doubt that a steady building up of the intensity of decompression effects (with their temporary weakening after experiments involving the development of caisson symptoms) is one of the main conditions for a rapid increase in the organism's resistance to decompression disturbances.

The results of experiments on a third dog (Timoshka) are shown in Figs. 24 and 25. As can be seen from Fig. 24, in the first period of the experiments, as the decompression effects were gradually intensified, the animal's resistance to them, as a general rule, increased rapidly and considerably. Subsequently,

after a few cases of especially intense decompression disturbances (the experiments labeled with the letter a), it decreased appreciably. Later (from the moment indicated in the figure by the arrow), we transfered the dog to daily experiments with a constant time of exposure to pressure. After six experiments with a 30-minute exposure, which terminated favorably, the dog's resistance to decompression disorders for some reason (such cases will be discussed later) decreased patently. In four experiments in succession (three with a 30-minute and one with a 25-minute exposure), the animal developed decompression disturbances. Another 14 experiments with a 25-minute exposure terminated without symptoms, after which the experiments were interrupted for a month and a half. The dog's resistance to decompression disturbances as a result of the interruption decreased considerably and even turned out to be at a lower level than at the very start of the investigations. /119

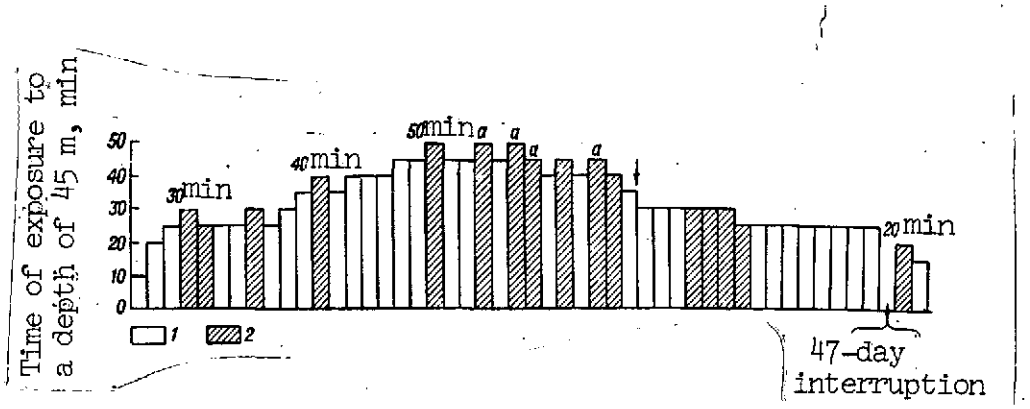

Fig. 24. Change in resistance to decompression incident to repeated effects in Timoshka. Experimental conditions and notation the same as in Fig. 21.

a — experiments with especially intense decompression symptoms

In the following experiments (Fig, 25), the resistance dropped rapidly to its initial level, after which daily experiments were begun with subliminal decompression effects. They continued for a month and a half. As can be seen from the figure, checking showed that with such a number of daily subliminal decompression effects, the organism's resistance to decompression disturbances increases considerably. /120

In the course of the investigation, other dogs, too, (Druzhok, Rex, Sedoy, Zor'ka), were subjected to the action of a

depth of 45 m and subsequent decompression. The experiments on these animals pursued special goals, but in their course a more or less considerable increase in resistance to decompression disturbances was noted in all the animals.

In four dogs earmarked for special investigations that do not enter into the present study, which were subsequently carried out by K. S. Yurova, we first determined the safe staying times for the animals at a depth of 100 m (a pressure of 11 psig) under the condition of rapid subsequent decompression to the surface.

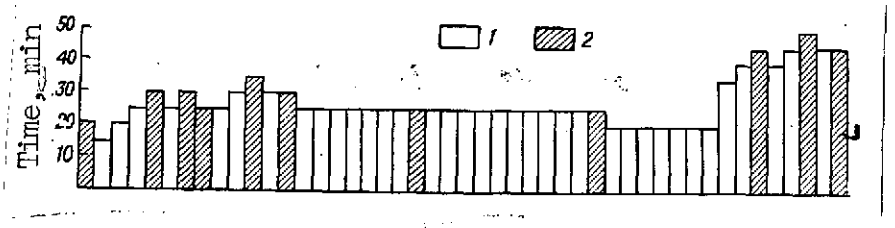

Fig. 25. Change in resistance to decompression incident to repeated effects in Timoshka. Continuation of experiments shown in Fig. 24. Notation the same as in Fig. 21.

At a depth of 100 m, as is well-known, for man and animals close to him in their resistance to decompression disturbances (dogs, rabbits), these times are reckoned in minutes, and the outcome of the effect can be decided in fractions of a minute. Despite such drastic conditions, in these experiments, too, an increase in the dogs' resistance to decompression disturbances was noted as the effects were repeated. In Fig. 26 are shown the results of preliminary experiments on Snezhok. As can be seen from the figure, when the order of applying the effects that we adopted (gradual intensification with temporary weakening after cases of decompression disorders) were adhered to, the dog's safe staying time at a depth of 100 m increased from 5 to 7 min.

Such an increase, as has just been pointed out, is very considerable for the conditions in question. In three other dogs, the increase in safe staying times as a result of the

experiments was even more considerable; we are not presenting these data inasmuch as the increase in resistance, which began in preliminary experiments, subsequently took place against a background of additional pharmacologic effects.

It was noted in experiments on six rabbits that if the experiments are begun at a depth of 100 m with exposures in the chamber that are known to be safe and if these exposures are gradually increased from time to time, then the animals will painlessly endure exposures that are fatal for fresh rabbits.

Evaluating the results of all of the experiments described in the present section, it should be taken into account that the conditions for them, as compared with the experiments involving determination of the safe supersaturation coefficient, were less strict. In determining the coefficient, owing to the long exposure of the animals to pressure, one and the same complete saturation of the organism with nitrogen was observed in every case at a certain depth, regardless of any dependence on these or those special features of the animals' condition and behavior in individual experiments. But in the experiments involving determination of safe staying times at medium and /121
greater depths, no such constancy of conditions could exist. Inasmuch as saturation here was incomplete, its degree at the moment of decompression for one and the same exposure time of the animal at the depth could to some extent change in connection with differences in the functional state of the animals in individual experiments. For these same reasons, as well as because the organism's saturation with nitrogen in the course of time occurs according to not a linear, but an exponential law, one and the same increase in the animals' exposure at a depth could correspond not always to one and the same increase in saturation. In a word, in determining safe staying times for animals at medium and greater depths, different attendant physiological moments could tell on the experiments to a greater extent than in determining the safe supersaturation coefficient. Obviously, that is precisely why, in the course of experiments involving exposure of the animals at a depth of 45 m, in all three dogs,(cf. Fig. 24-25), individual unexpected cases of decompression disturbances are noted against a background of repeated, quite satisfactorily transpiring, subliminal decompression effects, as well as periods of unexpected increase in the organism's resistance to these effects, periods lasting several days.

But even when it was not possible to maintain absolutely strictly the conditions determining the development of decompression disturbances, the fact of increase in the organism's resistance to these disturbances incident to repetition and gradual intensification of decompression effects emerged with complete distinctness in these experiments, too.

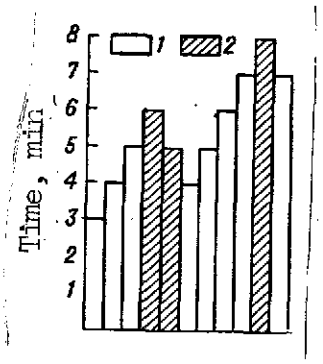

Fig. 26. Change in resistance to decompression incident to repeated effects in Snezhok. Overpressure in all experiments: 10 psig. Decompression: always single-step, at a rate of 1 atm every 15 sec. Notation the same as in Fig. 21.

These data, as well as the data on the increase in the safe supersaturation coefficient, were reconfirmed by a great number of experiments subsequently conducted on dogs by K. S. Yurova. Very striking confirmation of this fact had already been obtained in experiments on rabbits in the above-mentioned study by G. A. Lavrova. In her experiments, which involved a great deal of material, it was shown that when the animals' staying times at depths of 45 and 100 m were increased gradually, the corresponding safe exposures increased, as compared with the mean safe times, by a factor of 2 and more.

It is even somewhat /122 unexpected that rabbits, which are distinguished from other animals by the relative weakness of their organization, display such great adaptive resources in respect of decompression disorders.

Change in duration of safe stepped decompression methods incident to repetition of experiments

The third version of the experiments in which an increase in the organism's resistance to decompression disturbances was established, was experiments with stepped decompression after the animals' stay at a depth. In the course of these experiments, we determined in three dogs the shortest safe stepped decompression methods after the animals' stay at a depth of 45 m for 45 min and a depth of 80 m for 25 min.

The methods were patterned after the type that are used under the corresponding conditions in diving practice and incorporated into the rules of the diving service in 1959. The total safe times of stepped decompression for dogs were considerably shorter than the corresponding times for human beings. In decreasing the total duration of decompression, we proportionately reduced the staying times at all halts, thereby maintaining unchanged the character of the method.

Starting the experiments with methods that were known to be safe from the standpoint of duration, we reduced them from

experiment to experiment by 10-15 min.

The experiments showed that with repetition of the effects, the duration of safe stepped decompression methods decreases. Thus, after a 25-minute stay at a depth of 80 m, at first no decompression disorders appeared in Kudryash for a total decompression time of 64 min but appeared when it lasted 50 min. Subsequently, a 50-minute method became safe for the dog, and decompression symptoms arose only for 40-minute decompression.

In Manyunya, who was less stable to decompression disturbances, the duration of safe methods decreased from 98 to 85 min, while in Volchok, who was very resistant to decompression effects, it dropped from 40 to 30 min. The barograms of the corresponding methods are shown in Fig. 27.

These facts are important, in our opinion, for showing the possibility of enhancing the organism's resistance to decompression disturbances in respect of that type of decompression effect which is most often encountered in diving practice.

They call attention to themselves also for demonstrating in a particularly striking way the individual differences in the animals' resistance to decompression disorders. Indeed, the /123
difference in the duration of safe stepped decompression methods for the animals under consideration considerably exceeds those relative differences in the safe supersaturation coefficients for the safest staying times at the depth under the condition of subsequent single-step decompression which were noted in other animals and, in particular, in these same three dogs. Clearly, incident to stepped decompression from greater depths, the best conditions are created for the emergence of individual differences in resistance to decompression disturbances.

Experiments with a 45-minute stay at a depth of 45 m showed /124
that, under these conditions, in order to avert the development of decompression disturbances in dogs, there is no need to apply long stepped decompression methods analogous to those utilized in human practice. Only in the initial experiments was it necessary, in order to prevent decompression disorders, to apply decompression that was somewhat dragged out in time -- to 20-30 minutes for Kudryash and Manyunya and to 15 minutes for Volchok. But in subsequent experiments, the safe decompression time for the first two dogs decreased to 15 min, and for the last, to 5 min. Practically speaking, the matter came to a rapid transfer of the animals to the first halt (depth of 15-20 m) with subsequent decompression to the surface that was gradually slowed down, but was, on the whole, very short-lived. The corresponding decompression barograms are shown in Fig. 28. Consequently, as a result of the animals' increased resistance to decompression disturbances in the experiments under consideration, it became superfluous to apply any prolonged stepped decompression methods.

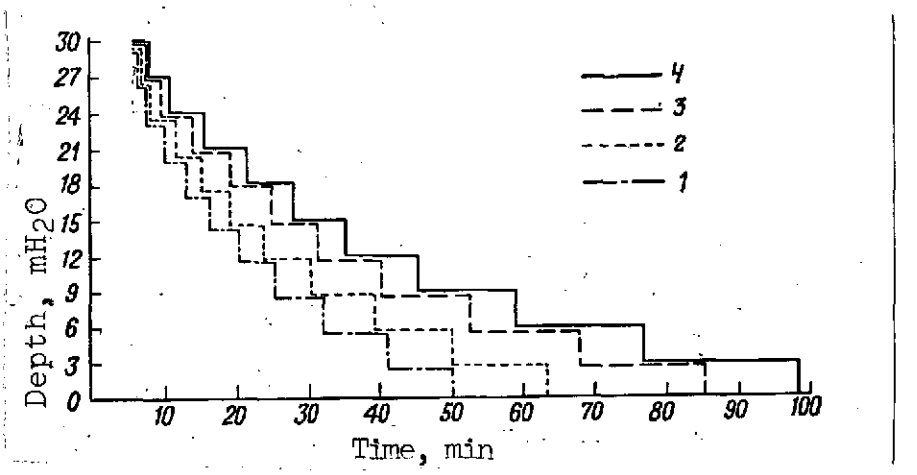

Fig. 27. Barograms of decompression methods utilized in experiments with dogs staying for 25 minutes at a depth of 80 m. Ascent time from 80 to 30 m and first halt (30 m): 5 min each. Ascent time from 30 m: 1 -- 50 min; 2 -- 64 min; 3 -- 85 min; 4 -- 98 min.

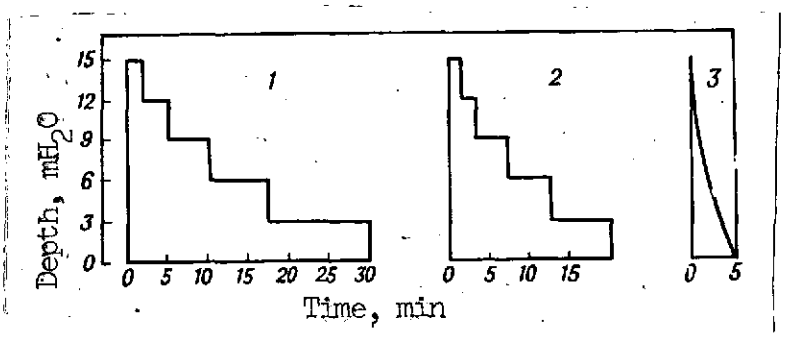

Fig. 28. Barogram of 30-minute (1), 20-minute (2) and 5-minute (3) decompression methods utilized in experiments with dogs staying 45 minutes at a depth of 45 m. Transition to first halt (15 m): 3 min.

Fig. 29. Ryzhik 3 min after decompression from under a pressure of 1.6 psig (four-hour exposure). Signs of decompression disturbances are absent.

Fig. 30. Ryzhik 2 min after ascent to an altitude of 4.5 km (ascent 5 min after decompression from under increased pressure). Same experimental conditions as in Fig. 29. Note the incipient signs of decompression disturbances: some inhibition of the dog and a lightening of the load on the right rear extremity.

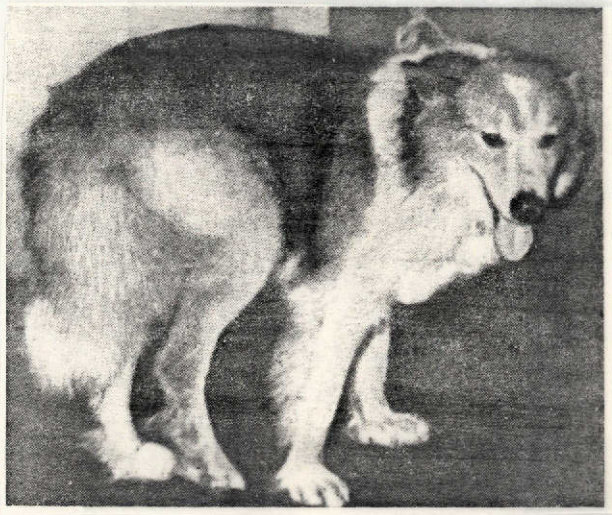

Fig. 31. Ryzhik. Fourth minute of stay at an altitude of 4.5 km. Same experimental conditions as in Fig. 29. Progressive signs of decompression disorders, the dog lifts its right rear extremity.

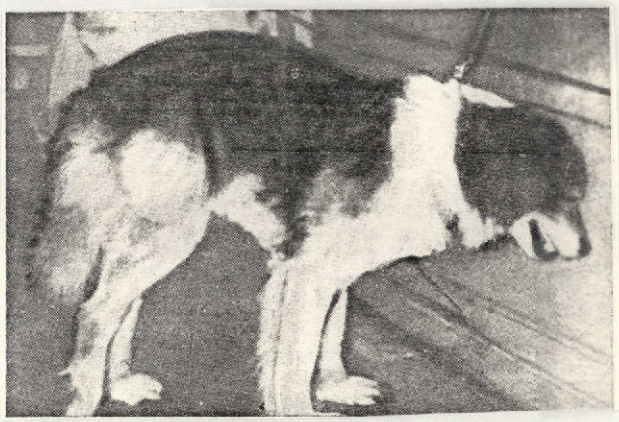

Fig. 32. Ryzhik in the same experiment. Sixth minute of stay at an altitude of 4.5 km. A pronounced decompression symptom of the paresis type has developed in the right rear extremity. The dog's general depression is conspicuous.

The results of all three versions of the experiments agree in showing that animal resistance to decompression disturbances increases regularly and considerably under the influence of repeated decompression effects of gradually increasing intensity. Consequently, to the question that was asked above, as to whether the organism possesses adaptive resources to the harmful effect of gas bubbles that are forming in its internal environment, the facts give a quite unambiguous affirmative answer. Despite the complete novelty and unusualness of the given factor, the organism proves able to counteract its noxious effect, adapts to it and enhances its resistance in a number of cases to such an extent that it comfortably endures decompression effects which, for the fresh organism, are fraught with very serious consequences or even certain death. The experiments showed that a rapid and considerable increase in resistance to decompression disturbances takes place in obedience to the general principles of conditioning of the organism to all effects and all types of activity, namely: gradual intensification of the effects with their temporary weakening in those cases where they begin to cause pronounced functional disturbances.

The data of Figs. 22 and 24 show that if we infringe this principle and do not temporarily weaken the effects after cases of pronounced functional disturbances, the resistance enhancement process slows down. And if the decompression disturbances arise repeatedly, several times in succession, or prove to be particularly intense, the organism's resistance decreases considerably and can be increased again only by reverting to deliberately weak effects and then increasing them with their former gradualness. Thus, repeated and even single-stage decompression effects /125 of excessive intensity have the opposite effect -- they lead to a reduction in the organism's resistance to decompression disturbances. All these facts were directly concerned in experiments undertaken by V. A. Aver'yanov [1964] for the special purpose of elucidating, incident to determination of the safe supersaturation coefficient, the optimal conditions for increasing the organism's resistance to decompression disturbances. In those cases where the author began determining the SSC from depths that were known to be dangerous, decreasing them from time to time, the decompression effects that had appeared in the dogs in the initial experiments arose subsequently, too, when the dangerous depths had already been traversed. In other words, the safe supersaturation coefficient, when determined by this method, proved to be lower than the usual mean figures. In those cases where the author gradually increased the depth but did not, after the appearance of decompression symptoms in the dogs, revert to lesser pressure, the resistance enhancement effect was incomplete. Those conditions to which we adhered in our experiments, on the other hand, i.e., gradual intensification of the effects with their temporary weakening after cases of decompression disorders, proved to be optimal.

It must be assumed that the organism's adaptation to decompression effects is affected also by another very important condition of all conditioning: frequency of these effects. Our data indicate that the intervals of 3-4 days that we adopted for decompression effects of increasing intensity ensure a conditioning effect that is good enough. But this does not mean that they were optimal; the question deserves special study.

The facts show that the organism's resistance to decompression disturbances, which increases as the effects are gradually intensified at the start of their application, will subsequently, if these effects are systematically repeated, keep to the higher level. This is not hindered even by periodically arising decompression disturbances, if they are not excessively intense and are liquidated in good time by recompression. Interrupting the effects for a month or a month and a half proves to be enough for the result of precedent conditioning to disappear and the resistance to drop to its initial low value.

But when the effects are resumed, resistance is very quickly restored, much more quickly than it was enhanced in the animal when it was first subjected to experimentation.

These experiments brought to light still another circumstance that deserves attention. They showed that the organism adapts not only to the intensity, but to the character of the decompression effects. Upon transition to each new version of the experiments with decompression in dogs, an additional, if one may say so, enhancement of resistance in respect of these new conditions is generally noted. Thus, for example, dogs /126 which had been in long systematic experiments involving determination of the safe supersaturation coefficient and in which resistance to decompression disturbances had been established at a higher level, upon transition to decompression effects of a different character (determination of safe staying times at great depths, determination of safe multi-step decompression methods), more enduring from the very beginning as they are than freshly-taken animals, in some experiments of the next series already display an increase in resistance in respect of these new conditions.

Thus, superimposed on the general increase in resistance to decompression disturbances, the best measure of which is obviously the safe supersaturation coefficient, is an additional conditioning effect connected with the specific character of the applied decompression effects.

To be stressed, finally, is that even when an absolutely strict system of applying decompression effects is adhered to, resistance to decompression effects may fluctuate. In the light of all the facts at hand, such fluctuations are not only not surprising, but even inevitable. Indeed, as soon as we have

to do with the organism's active physiological adaptation to caisson phenomena, there can be no doubt that the different changes brought about for one reason or another in the organism's general condition, cannot but tell on its adaptive forces.

The results of these experiments show the complete untenability of the idea that the organism's sensitivity to "caisson disease" changes but little, that its ability to cope with certain degrees of supersaturation is constant and relatively stable (in particular, the assertion of I. I. Savichev and A. A. Bukharin that the "limit" in individuals is constant). These wrong ideas must be discarded once and for all because they conceal a double jeopardy. In the first place, they blunt the attention of physicians in charge of the work and condition of divers, which can be of decisive significance for the outcome of their dive and, secondly, such ideas cut off the promise of conditioning and increasing the resistance of divers to that factor which constitutes their gravest occupational hazard.

What are the physiological reactions on which the increase in the organism's resistance to decompression disturbances is based, is a question that will be discussed in detail in a later section of this book. Here it is only necessary to note that, as a rule, the resistance enhancement process, which is distinguished by the absence of manifest pathologic disturbances (symptoms of the bends, profound general depression and poor condition of the animals), keeps the changes in respiration and heart action that are specific to incipient caisson phenomena, /127 somewhat altered in character though they may be.

Lastly, we must dwell on what conclusions about experimental study of decompression disorders ought to be drawn from the material we have presented.

The facts adduced in the present study and those being currently gathered by our teammates leave no doubt that in the course of chronic experiments with decompression effects, the increase in the organism's resistance to decompression disorders becomes ingrained. This circumstance hampers and retards solution of a number of problems concerning decompression (the influence of different additional effects on the development of decompression disturbances, the comparative action of different gaseous mixtures and many others). But to eliminate it is impossible, and it is up to the researcher to know how to vary the intensity of decompression effects with due regard for the increase in the animals' resistance to them and to ensure his fundamental results by a sufficient number of repeated control experiments. Otherwise very shaky conclusions may be drawn.

The results of the experiments described in the present chapter lead to the following conclusions.

Incident to repeated, gradually intensifying decompression effects (after a stay under a high pressure), the organism's resistance to decompression disturbances increases considerably. This bears witness to the fact that the organism possesses adaptive resources in respect of so extraordinary a factor as free gas bubbles in its internal environment.

The organism's resistance to decompression disturbances that have been increased under the indicated conditions keeps to the higher level provided the organism is systematically subjected to decompression effects of not excessive intensity. Upon interrupting these effects for one to one and a half months, resistance drops to its initial level, but can be rapidly restored by reconditioning.

As a result of particularly intense decompression disturbances, whether they arise repeatedly or but once, the organism's resistance to them decreases considerably.

The organism's resistance to decompression disorders undergoes fluctuations apart from decompression effects, too. The idea of its constancy is profoundly erroneous and must be discarded.

The considerable changes that take place in the organism's resistance to decompression disturbances under the influence of decompression effects themselves, must be taken into account in carrying out any experimental study devoted to this problem.

CHAPTER 8 /128

LATENT FORMS OF CAISSON PHENOMENA AND THEIR INDUCEMENT BY MEANS OF "ASCENT TO AN ALTITUDE"

The foregoing facts taken together compel the assumption that the caisson phenomena arising in the organism incident to decompression at first take a latent and veiled course and lead to manifest functional disturbances and development of specific decompression symptoms only after attaining a certain intensity. This assumption was suggested first of all by observations of animals after decompression. They showed clearly that the first and most abiding consequences of decompression are changes in respiration, heart action and the animal's general condition. It was natural to regard these not very striking physiological shifts, which always precede the development of manifest functional disturbances and specific decompression symptoms, as a consequence of the process of gas-bubble formation that has begun in the organism but has not yet developed into typical and pronounced functional disorders.

Further, the assumption of latently proceeding caisson phenomena ensued directly from data about the values of the physical coefficient of maximum permissible supersaturation of the blood, which determines the beginning of gas formation in it, and of the coefficient of safe supersaturation of the organism, which determines the development of manifest decompression disturbances. It was natural to assume that the regular and considerable discontinuity between the physical coefficient and the safe supersaturation coefficient corresponds to that period in the development of caisson phenomena when they do not yet cause typical pathologic symptoms.

Finally, convincing evidence for the correctness of the assumption of latent and little noticeable forms of caisson phenomena was furnished by data on the increase in resistance of animals to decompression disturbances. There can be no doubt that this increase in resistance can take place only on the basis of the organism's interaction with the gas bubbles that are being formed in its internal environment. Decompression by itself, if it does not lead to formation of gas bubbles, cannot be a stimulus for the development of adaptive defense reactions that ensure an increase in the organism's resistance to decompression disturbances. It must therefore be assumed that at a certain moment in the course of repeated, gradually intensifying decompression effects, gas bubbles always arise in the organism, but that they lead to manifest decompression disturbances in the course of such conditioning only in individual cases, while in all other cases, they cause no such disturbances, but do elicit the beginning of adaptive reactions.

Thus, the assumption of latent forms of caisson phenomena followed logically from existing data, and it had to be subjected to direct experimental checking. /129

It should be noted that the literature contains allusions by different authors [Harvey, 1951; Nimms, 1951] to so-called silent gas bubbles. These allusions, however, besides being to a considerable extent of a hypothetical nature, are confined only to discussion of a narrow question: can there or can there not exist in the organism gas bubbles that do not cause specific decompression symptoms? In these allusions there is no question of latent and little noticeable forms of caisson phenomena as an obligatory link in the developmental mechanism of decompression disturbances.

To study this question and check the above-mentioned assumptions, we employed the following method of procedure. At different times after decompression from under increased pressure, in the absence of decompression disturbances in the animals,

we "raised" them to an "altitude,"[9] i.e., subjected them to the effect of a rarefied atmosphere. In so doing, we used such degrees of rarefaction as were known to be in themselves unable to cause decompression disorders or lead to acute hypoxemic phenomena.

At first we undertook special experiments to induce latent forms of decompression disturbances by the above-mentioned method. These experiments were conducted jointly with A. A. Savich on nine dogs in which repeated determinations of the coefficient of permissible supersaturation of the organism with nitrogen had been carried out up to that time. The animals were placed in the chamber under a pressure known to be less than that which, in experiments involving determination of the SIC (safe immersion coefficient), caused decompression disturbances. For individual animals in different experiments the pressure varied from 1.2 to 2.6 psig. After a four-hour exposure to pressure, the dogs were subjected to rapid decompression to the surface, after which constant watch was kept of them. Under ordinary conditions, no decompression disturbances (symptoms of the bends) developed in the animals. At different times after decompression (from 15 min to 1 hour 45 min) the dogs were subjected to the action of rarefaction in the vacuum chamber (ascent to an altitude of 4.5 km at a rate of 1 km every 30 sec). The experimenter was either in the chamber together with the animal, or observed it through a porthole. If the dogs developed manifest decompression disorders, they were immediately returned to the surface; if they did not, they were kept at an altitude of 4.5 km for 10 min.

After decompression before ascent to an altitude, in no case were symptoms of the bends type noted in the animals. Noted in a number of experiments were only slight panting and some general inhibition of the dogs. /130

In almost half of all the experiments (25 out of 57), ascents to an altitude caused typical caisson symptoms to appear in the dogs. All cases of development of these symptoms incident to rarefaction of the atmosphere can be clearly divided into two categories.

The first category (it is of particular interest) consists of cases where the caisson symptoms developed in the course of the ascent itself. Usually these symptoms appeared at once in

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[9] In what follows the terms "ascent," "altitude," and "descent" -- which are customary in working with vacuum pressure-chambers -- will be used without quotation marks.

a pronounced form: the dog raised an extremity either right from the start of the ascent or at an altitude of 2-3 km. Sometimes it was possible to spot the first signs of caisson symptoms immediately from the start of rarefaction: the dog began to stamp its paws, lightened the load on one or another extremity, began to be wary of it. With a further ascent, the symptoms very quickly became manifestly pronounced: the dog raised the affected paw, fell into profound anxiety, which was apparently connected with acute pain, and began to whine, hopping on three legs. In contrast to the cases described earlier, events here developed rapidly and violently; as the ascent continued, the suddenly arising symptoms progressed before our eyes, literally in keeping with the altimeter pointer, and often attained such an intensity even before the altitude of 4.5 km that we had to discontinue the ascent immediately and increase the pressure in the chamber.

The second category consists of cases where the symptoms of the bends develop at different times after the altitude has been reached. The ascent itself, i.e., the period of rarefaction of the air in the chamber, did not cause any signs of functional disturbances involving the extremities, not even incipient ones. On the occasion of the first stay at the altitude, too, these signs were completely absent. They appeared only after a lapse of 2-5 min, gradually intensified and changed into manifestly pronounced caisson symptoms. Generally speaking, the character of the symptoms, and the dynamics and tempo of their development, were in all cases quite the same as incident to the onset of decompression disturbances under ordinary conditions after emergence from under a pressure in excess of the dog's SSC. Figures 29-32 show one of the cases of such gradual development of caisson symptoms in Ryzhik at an altitude.

Incident to immediate descent and return of the animals to ordinary pressure, the decompression symptoms involving the extremities in all cases vanished without a trace. Table 5 shows the results of all 57 experiments.

Gradual development of the symptoms of the bends after reaching the altitude took place in 13 cases, and their rapid development in the course of the ascent, in 12. It can be seen from the table that both categories of disturbances were noted in the animals after a stay at different depths and incident to different intervals between decompression and ascent. A circumstance that must be stressed is that the symptoms of the bends, which developed rapidly in the course of the ascent itself, /132 seldom arose in dogs after their stay at depths that were considerably less than the maximum permissible ones. The duration of the interval between decompression and ascent, as Table 5 shows, is of significance for the development of symptoms of one kind or another. The majority of cases of gradual development of symptoms at an altitude were observed for short intervals

and were not encountered after the animals' stay under ordinary conditions for more than an hour. But there were cases of rapid development of disturbances in the course of the ascent after short intervals, as well as intervals as long as one hour 45 min.

It should be particularly stressed that the ascents to an altitude induced not only the development of bends symptoms: they brought out and intensified such earlier and regular signs of caisson phenomena as panting and the animals' general inhibition and depression. The inducement of these signs was even a considerably more regular result of the ascents than that of the bends symptoms. Only in experiments with long intervals between decompression and ascent (more than one hour 20 min - one hour 30 min) did rarefaction not lead to pronounced panting and depression of the dogs. But in all the other experiments, incident to the ascent these phenomena were striking. In a number of experiments, when, after return from a shallow depth, it was difficult to notice changes in the dogs' breathing and general condition under ordinary circumstances, rarefaction from the very beginning brought out panting and depression of the animal, which progressed rapidly with altitude. In those cases where these phenomena were noticeable even under ordinary conditions, after return from the depth, incident to the ascent they intensified before our eyes and at the altitude attained a very high intensity. The development of pronounced panting and general depression of the animals accompanied also all those ascents to an altitude in which the appearance of bends symptoms was noted. In all of the enumerated cases, so intense were panting and general depression of the dogs incident to ascent to an altitude that it was impossible to explain them by that slight hypoxemia which arises at altitudes of several kilometers. Control experiments with ascents of dogs to an altitude of 4.5 km showed, in conformity with generally known facts, that incident to the given degree of rarefaction of the atmosphere, the dogs developed extremely slight and often almost imperceptible changes in their respiration and general condition which do not bear any comparison with those which take place incident to an ascent of animals that have until that time been under increased pressure. In a number of experiments in which caisson phenomena were induced by an ascent of animals that had been under increased pressure, at the altitude general functional disorders in the form of very acute depression, weakening of the general musculature tonus, uncoordinated movements, profound inhibition of reflexes and very hard panting emerged to the foreground, acquired a threatening character and compelled a descent to be undertaken at once, independently of the presence or absence of the bends symptoms. /135

In evaluating the data obtained, it is necessary first of all to dwell on cases of bends symptoms that develop rapidly in the course of ascent to an altitude in animals that

TABLE 5. DECOMPRESSION SYMPTOMS IN DOGS INCIDENT TO ASCENTS TO AN ALTITUDE OF 4.5 km AFTER DECOMPRESSION FROM UNDER INCREASED PRESSURE

Experiment number	Depth, m	Interval between decompression and ascent, hours and min.	Time of onset of symptoms
Zor'ka (28)*			
1	18	1.20	–
2	11	0.15	–
3	20	0.15	–
4	18	1.40	–
5	22	0.15	–
6	24	1.20	–
7	26	0.15	–
8	26	0.15	7th min. at alt.
9	26	1.40	Incident to ascnt.
10	26	0.30	2nd min.at alt.
11	26	0.15	–
12	26	1.30	–
Rex (24)			
1	16	1	–
2	18	0.25	5th min.at alt.
3	18	0.40	3rd min.at alt.
4	18	1.20	Incident to ascnt.
5	19	1.40	–
Sokol (26)			
1	18	1.00	–
Valet (22)			
1	16	1.00	–
Mishka (22)			
1	14	0.45	–
2	14	1.20	–
3	16	0.15	5th min.at alt.
4	16	0.30	3rd min.at alt.
5	18	0.25	Incident to ascnt.
6	18	0.30	–
7	18	1.30	–
8	18	1.30	–
9	18	1.40	Incident to ascnt.
10	19	0.40	–
11	22	0.15	3rd min.at alt.
Druzhok (26)			
1	12	0.15	–
2	18	0.45	6th min.at altitude
3	20	0.30	Incident to ascent
4	22	0.15	–
5	22	1.00	7th min. at altitude
Sedoy (18)			
1	16	0.45	–
2	18	0.45	–
3	18	0.15	–
Bogatyr' (22)			
1	14	0.45	Incident to ascent
2	14	1.40	–
3	16	0.15	Incident to ascent
4	16	0.45	Same
5	18	0.15	4th min. at altitude
6	18	1.40	Incident to ascent
7	20	0.25	–
8	20	0.30	Incident to ascent
9	20	0.30	Same
10	20	1.00	–
11	20	2.00	–
Ryzhik (22)			
1	14	0.30	–
2	14	0.45	Incident to ascent
3	14	1.15	–
4	16	0.16	–
5	16	0.16	3rd min. at altitude
6	18	0.15	5th min. at altitude
7	18	1.30	–
8	18	0.45	2nd min. at altitude

* Depth corresponding to the animal's SSC (in m).

had been under safe increased pressure. These facts can be explained only by the circumstance that even before the ascent, despite the complete absence of any outward signs whatsoever of typical functional disturbances involving the extremities, there already existed, in certain places of the organism, gas bubbles that had been formed. The sudden and immediate appearance of symptoms that developed incident to rarefaction in these cases can be explained only by expansion and increased pressure of the gases in a bubble that had been formed earlier; in other words, intensification of those mechanical effects owing to which the bubble causes painful sensations by acting on nerve endings.

In the case under consideration, therefore, it is a question of the presence of "silent" gas bubbles in those places where they develop their physiological effects, eliciting the typical symptoms involving the extremities[10]. It is significant that these bubbles -- potential causative agents of caisson symptoms -- were already formed in individual dogs (Ryzhik, Mishka) after a pressure of 1.6-1.4 psig, i.e., 0.6-0.8 atm lower than the maximum permissible value for the animals' safe supersaturation coefficient. These facts indicate that between the start of gas formation and the onset of typical caisson symptoms there is a considerable gap; in other words, that caisson phenomena at first proceed latently and lead to development of typical functional disturbances only after attaining a certain intensity.

This second category of cases, in which caisson symptoms develop slowly and gradually after reaching a high altitude, is apparently connected with formation of gas bubbles already under these conditions. Here it is conceivable that up to the ascent, either there were no gas bubbles in these places at all or only very fine ones, far from the critical size. Additional decompression, by increasing the degree of supersaturation of the blood and tissues, must of course be conducive to both formation of new gas bubbles and gradual increase in size of already existing fine gas seeds. The circumstance that cases of gradual development of bends symptoms at an altitude coincides mainly with earlier periods after decompression is quite understandable: rarefaction is a distinctive measure of the organism's supersaturation with nitrogen in the postdecompression period. Supersaturation decreases in the course of time, and in later periods, the danger of formation of new gas bubbles abates. Here one might expect only decompression disturbances connected /136 with gas bubbles that were formed earlier but did not put in an appearance.

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[10] The question of the localization of these gas bubbles will be discussed below.

As far as the data concerning inducement of such symptoms as panting and the animals' general depression by means of an ascent to an altitude is concerned, these data seem to us no less significant than inducement of the bends symptoms. They indicate that the latent forms of caisson phenomena cannot be reduced to mere existence in the organism of "silent" gas bubbles situated in some typical and, one must suppose, limited regions of the extremities, from where they can cause development of the bends symptoms. The onset of panting and of the animals' general depression, which coincides with the start of rarefaction, as well as the rapid intensification of these symptoms in the course of the ascent, point to the fact that the latent, outwardly almost imperceptible period of caisson aeroembolism is a regular and obligatory phase in the development of decompression disturbances.

Subsequently, we began systematically to use this method of inducing decompression disorders in our investigations. This had a twofold purpose: firstly, with the aid of this method it was possible to evaluate the degree of safety of the decompression effects being used, as well as the efficaciousness of therapeutic recompression, and, secondly, by conducting tests with ascents to a high altitude under different conditions, it was possible to count on getting additional material about latent forms of caisson phenomena. The ascents to an altitude were executed one hour and one hour, 30 min and two hours after decompression effects connected with prior exposure to increased pressure only in those cases where in the course of the indicated periods, no decompression disturbances developed in the animals and it was clear that the danger of their development under ordinary conditions was past. Only sometimes did we have recourse to ascents to an altitude in other periods -- in order to bring out the presence of decompression disturbances of the bends type, when at the surface there had been only unclear hints of symptoms that did not intensity with the passage of time.

After therapeutic recompression, which was generally carried out until complete liquidation of decompression symptoms, the animals were raised to an altitude immediately upon their emergence from the compression chamber.

In all cases, we adhered to one and the same ascent schema: rarefaction up to an altitude of 4.5 km at a rate of 1 km every 30 sec, a five-minute stay at this altitude, ascent to 6 km at the same rate, a 30-second stay there and descent to the ground. In the event of decompression disturbances (bends symptoms or the dog's poor general condition), ascent was interrupted as soon as these disturbances became absolutely manifest and difficult to bear for the animal.

Below, we present the summary results of experiments on nine dogs involving ascents to an altitude one hour, one hour 30 min and two hours after decompression from under increased pressure (different values), decompression which caused no specific signs of decompression disorders in the course of the indicated times under surface conditions: /137

Number of experiments	Development of symptoms			
	Total number of symptoms	Rapid, in the course of ascent	Gradual, at the altitude	
			4.5 km	6 km
67	47	25	9	13

The data that we presented earlier, in conformity with information in the literature, bear witness to the fact that the development of bends symptoms in dogs under ordinary conditions generally takes place in the course of the first 30-40 min after decompression. The appearance of these symptoms after one hour takes place in solitary cases, after an hour and a half is a still rarer exception, and after two hours is practically not encountered. Consequently, inasmuch as the decompression effects caused no typical symptoms in any of these animals in the course of the indicated times, we are entitled to conclude that these effects terminated favorably. But ascents to an altitude resulted in a rapid and acute onset of symptoms in more than 1/3 of all cases, which points to the fact that in all of these dogs, gas bubbles of typical localization had already been formed which did not manifest themselves under the ordinary conditions of their action. In nine cases, a gradual development of symptoms took place at an altitude of 4.5 km, which points to the fact that up to the time of ascent, the animals in question were straddling the boundary of maximum permissible supersaturation and danger of decompression disturbances.

Noteworthy are data on three dogs incident to ascents to an altitude two hours and two and a half hours after rapid decompression from under a pressure of 4.5 psig, a decompression that entailed no specific decompression symptoms.

Dog	Number of experiments	Development of symptoms	
		Rapid, in the course of ascent	Gradual, at the altitude
Volchok	5	2	1
Kudryash	10	6	-
Timoshka	8	5	-
In all	23	13	1

During these times, the animals were undoubtedly already out of any danger of developing decompression disturbances under ordinary conditions. In the investigations of a number of authors, two hours of observation of the dog after decompression is assumed to be absolutely sufficient to conclude whether the /138 effect did or did not lead to decompression disorders, and on expiration of this time, the experimenter leads the dog back to the vivarium with, as they say, an easy conscience, being quite convinced of the absence of caisson phenomena in it.

The figures that we have presented, however, indicate that in our case, in 13 experiments out of 23, local gas bubbles were detected to have been formed during these times in dogs that showed decidedly no functional disturbances on the ground. Gradual development of symptoms at an altitude at these late times took place only in one experiment. This bears witness to the fact that for most of the animals, the period of dangerous degrees of total supersaturation of the organism with nitrogen was already past.

In four experiments carried out on these same dogs, experiments which do not enter into the data that have just been presented, the bends symptoms that developed soon after return from a depth were not distinguished by any great intensity, and they vanished spontaneously and completely without leaving any trace in three cases at the end of one hour, and in the other, at the end of an hour and a half. Incident to ascents to an altitude effected two hours and, in one case, two hours 30 min after decompression (i.e., 1/2-one hour after liquidation of the symptoms), decompression disturbances in the same extremities appeared in an acute form with the very first degrees of rarefaction; consequently, the bubbles that caused the symptoms had not yet been liquidated, but had apparently only been reduced in size.

Below are presented the results obtained on two dogs when using two-step decompression methods from a depth of 60 m (pressure of 6 psig) with a 30-minute exposure. In the experiments, we varied the depth of the intermediate halt (from 12 to 18 m) and its duration (from 30 to 60 min). In the event the dogs developed no decompression symptoms in one hour 30 min after decompression, ascents to an altitude were undertaken. The experiments showed the following:

Dog	Number of experiments	Development of symptoms	
		Rapid, in the course of ascent	Gradual, at the altitude
Sokol	5	3	-
Sedoy	6	3	-
In all	11	6	-

Thus, in six out of 11 experiments indicating, it would seem that safe parameters had been found for the two-step decompression method, the dogs had latent gas bubbles.

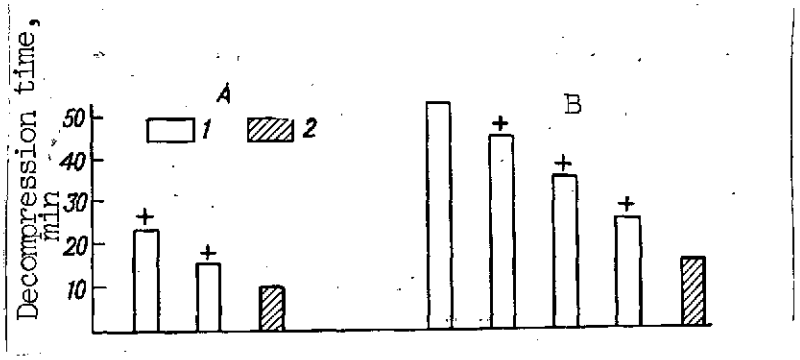

Fig. 33. Check for the presence of latent gas bubbles in the organism after a 45-minute stay of Volchok (A) and Manyunya (B) under a pressure of 4.5 psig and stepped decompression of different durations.

1 -- without decompression disturbances; 2 -- with disturbances; the + sign designates experiments in which the presence of bubbles was detected incident to ascents.

Worthy of particular attention is checking on the safety of multi-stepped decompression methods patterned after those employed in diving practice. The results of such a check-up /139 are shown in Figs. 33 and 34. They show that gas bubbles that cause symptoms of the bends are formed when using multi-stepped methods that exceed by far in their duration the shortest or, as they say, critically drastic safe methods. Thus, in the case of a depth of 45 m, ascents to an altitude brought out the presence of latent gas bubbles in Volchok after 23 minutes of

decompression and in Manyunya, after 45 minutes. But the duration of safe methods, to judge by the development of symptoms under ordinary conditions, amounted for the first dog to 15 min, and for the second, to 25 min. After return /140

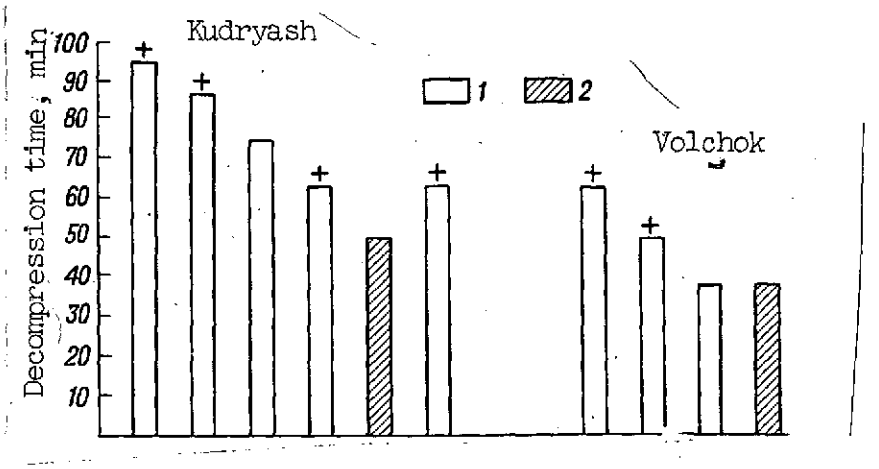

Fig. 34. Checking for the presence of latent gas bubbles in the organism after a 25-minute stay of Kudryash and Volchok under a pressure of 8 psig and stepped decompression of different durations. Same notation as in Fig. 33.

from a depth of 80 m (25-minute exposure), latent gas bubbles were detected in Kudryash incident to ascent to an altitude after 95 minutes of decompression, whereas, to judge by the symptoms, a 63-minute method proved to be safe. The presence of symptomless bubbles in Volchok was detected after 63 minutes of decompression, whereas a 38-minute method proved to be safe. These data directly suggest the idea that the decompression methods used in diving practice by no means always prevent the formation of local gas bubbles in the organism and that they are regarded as safe only because a considerable gap exists between the formation of bubbles and the appearance of symptoms.

The results of checking the efficaciousness of therapeutic recompression are shown in Table 6. It includes experiments with different therapeutic methods, comparison of whose efficaciousness will later be the subject of special consideration. The only criterion by which the experiments in the table were selected is whether using therapeutic recompression in them led to complete liquidation of existing decompression disturbances in the dogs. The data of the table show that the symptoms reappeared in an acute form in the course of ascent to an altitude in 26 out of 77 cases.

TABLE 6. RESULTS OF CHECKING THE EFFICACIOUSNESS OF THERAPEUTIC RECOMPRESSION BY MEANS OF ASCENTS TO AN ALTITUDE

Dog	Number of experiments with ascents to altitude after outwardly complete therapeutic effect	Rapid development of former symptoms in an acute form in the course of ascent					Unclear incipient signs in the course of ascent	Gradual development of symptoms at an altitude	Emergence of new symptoms
		In all cases	including:						
			at start of ascent	incident to ascent to:					
				2 km	3 km	4.5 km			
Ryzhik	12	3	—	1	—	2	—	—	—
Druzhok	4	2	—	—	1	1	—	—	—
Mishka	13	5	1	1	1	2	2	2	—
Sedoy	9	5	1	1	2	1	2	2	—
Bogatyr'	15	3	2	—	—	1	1	—	2
Sokol	3	1	—	1	—	—	1	—	—
Zor'ka	1	—	—	—	—	—	—	1	—
Rex	1	—	—	—	—	—	—	1	—
Volchok	4	2	—	—	1	1	—	—	—
Kudryash	7	4	—	1	1	2	—	—	—
Timoshka	8	1	—	1	—	—	—	—	—
In all	77	26	4	6	6	10	6	6	2

These symptoms appeared in every dog precisely in that /141
extremity which suffered after return from a depth, were at first quite similar in character to the symptoms existing before recompression but, on continuing the ascent, acquired greater intensity. These data allow no room for doubt that in all of these cases, therapeutic recompression did not lead to liquidation of the bubbles that cause symptoms of the bends, but merely reduced them to a subliminal size. It is safe to assume that the lower the altitude at which the symptoms reappear, the greater the size of the gas bubbles that are retained in a latent form. The data of the table show that in most cases, the symptoms developed at low altitudes; this bears witness to the fact that the bubbles were still rather large and that therapeutic decompression reduced them to a size differing but little from threshold values. Besides the 26 experiments in which the

symptoms developed in an acute form incident to ascent to an altitude, in six experiments, incipient and not quite clear signs of functional disturbances occurred incident to rarefaction, and in another six, gradual development of bends symptoms involving extremities that had suffered earlier. Obviously, these latter cases must be explained by the persistence of local supersaturation in places where bubbles formerly existed and, perhaps, also by the persistence there of very fine bubbles that need time to grow to physiologically significant sizes. Finally, two cases in the table occupy a place apart, when disturbances of the bends type develop at an altitude gradually, but not in that extremity in which they had existed up to therapeutic recompression, but in another extremity that was unharmed earlier (Bogatyr'). These cases mean that the therapeutic methods used in them can themselves threaten the organism with decompression disorders.

Thus, the experiments showed that the efficaciousness of therapeutic recompression often proved to be somewhat deceptive. Therapeutic recompression eliminates decompression symptoms all right, but very often does not completely liquidate their cause: gas bubbles. At the end of therapeutic re- and decompression, the organism turns out to be on the verge of redeveloping decompression disturbances. The "full curative effect" that researchers usually talk about is far from always being really full, and the "cure" itself, which is regarded as radical when the symptoms are liquidated, has in most cases a palliative character.

In our opinion, these facts merit serious consideration in connection with improving the therapeutic methods employed in diving practice, all the more as many of the methods that we employed were designed along the same principles as those used for human beings. The most significant fact to emerge from these experiments involving inducement of decompression phenomena by means of ascents to an altitude is that rarefaction in all cases (when the time after return from under increased pressure is not too long) brings out clear signs of an aeroembolic process in the form of panting and a change in the organism's general condition. Whence it follows that aeroembolism is a regular and obligatory initial stage of caisson phenomena. Strictly speaking, the expression "latent caisson phenomena" is not quite appropriate for incipient postdecompression aeroembolism; aeroembolism, as was said above, even without the additional effect of rarefaction, generally finds its, albeit not always clear, expression in panting, changes in cardiovascular activity and inhibition of the animals. But ascent to an altitude makes these phenomena striking, the high speed with which they develop and progress in the course of the ascent bearing witness to the fact that formation of gas bubbles in the blood was already taking place under normal pressure. Development of panting that

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is inappropriate for the low degrees of rarefaction at which it arises is, as has already been noted, a considerably more constant manifestation than development of symptoms of the bends. It must therefore be assumed that the gas bubbles that cause these symptoms are formed after a stay under increased pressure at a certain stage in the development of postdecompression aeroembolism. This conclusion does not conflict with the assumption of intracellular localization of the gas bubbles that determine the onset of symptoms of the bends.

By retarding and impairing the blood flow, and so the desaturation of the tissues, too, incipient aeroembolic phenomena must be a precondition for intracellular formation of gas bubbles. The formation of latent and very persistent gas bubbles in the organism in those places where they can cause painful symptoms involving the extremities is indubitably an important fact. But, as it seems to us, this fact is overshadowed by incipient postdecompression aeroembolism, which often appears in a masked form or manifests itself in such unspecific symptoms that they escape the researcher's attention. The pathogenesis of decompression disorders incident to decompression from under increased pressure can in no way be reduced to the specific symptoms of the bends if for no other reason than that they develop in far from all animals. It would be more correct to regard these symptoms in man and those animals in which they appear as a very clear and striking indication that caisson phenomena in the organism have attained a certain intensity and are going over to a phase of pronounced functional disturbances. But of incomparably greater significance for the development of caisson phenomena as a whole is decompression aeroembolism, all the more as it is connected, as will be shown below, with defense reactions of the organism that determine its resistance to decompression disturbances. /143

The factual material that has been presented above permits the following conclusion to be drawn.

Ascents of animals to an altitude after decompression from under an increased pressure that is known to be safe is accompanied by the development of pronounced panting and general depression, which bears witness to the fact that aeroembolic phenomena took place even before the ascent. The typical caisson symptoms involving the extremities that develop in a number of experiments can be either one of two kinds. In some cases, they develop immediately after the start of the ascent, and in others, at different times after reaching the altitude.

After return from depths that are known to be safe, gas bubbles -- potential causative agents of bends symptoms in the absence of any functional disturbances whatsoever involving the extremities -- can arise in the organism and persist for a long time.

In almost half of all cases, the therapeutic recompression that is applied to dogs with pronounced bends symptoms and that liquidate these symptoms utterly, does not lead to complete destruction of the gas bubbles that cause these symptoms.

The method of inducement of caisson phenomena by means of ascents to an altitude is a criterion for the presence of "silent" gas bubbles in the organism and a measure of its supersaturation with nitrogen. It shows promise of further experimental and, possibly, practical development.

CHAPTER 9

ROLE OF AEROEMBOLIC PHENOMENA IN THE DEVELOPMENT OF DECOMPRESSION DISORDERS

The foregoing material bears witness to the fact that caisson phenomena in the organism begin with formation of gas bubbles in the venous blood and that aeroembolism by itself determines the entire subsequent course of the development of general decompression disturbances. This conclusion, in spite of being in complete agreement with the data of A. P. Brestkin [1952] about the lesser ability of the blood, as compared with the tissues, to retain in solution the nitrogen with which they are supersaturated, could not be accepted as definitive only on the basis of outward manifestations of decompression disorders. In order to ascribe to it the force of an incontestible conclusion, it was necessary to turn to crucial experiments and immediate study of aeroembolic phenomena themselves, with simultaneous accurate recording of the reactions evoked in the respiratory /144 and cardiovascular systems by aeroembolism. The present chapter gives an account of the results of such experiments.

Decompression aeroembolism

The crucial experiments involving study of decompression aeroembolism mainly in dogs were carried out in two versions. In the first, in which serious forms of caisson phenomena were investigated, at the same time that we recorded respiration and blood pressure on a kymograph, we kept a visual watch for gas formation in the vessels. These experiments, which were conducted jointly with I. I. Vavilov, were undertaken for the purpose of first obtaining a general impression of the pattern of pronounced decompression disorders and the role of aeroembolic phenomena in their development. In the second version of the experiments, we used decompression effects of different intensities ranging from very weak to fatal and, as in the first version, recorded the changes in respiration and cardiovascular activity; but we

checked for gas formation in the blood with the aid of gas traps. These experiments were aimed at more accurate determination of the beginning of gas formation in the blood, its intensity later on and comparison of these data with the changes in respiration and blood circulation.

The experiments of the first type were conducted on 18 dogs under morphine-urethane narcosis. The morphine dose, which was injected subcutaneously in a 2% solution, amounted to 0.01 g/1 kg weight.

The urethane dose, which was injected in fractional portions intramuscularly in a 10 or 20% solution, was determined by the required deepness of the narcosis (absence of corneal and pain reflexes while keeping the normal character of respiration and blood-pressure level); it varied from 0.4 to 0.8 g/1 kg weight.

The animals were subjected to a tracheotomy and a gastrotomy (into the stomach was introduced a wide metal tube for the purpose of decreasing the effects of meteorism incident to decompression); the recording of blood pressure was arranged for the femoral artery, and sections of the skin were separated on the stomach, thorax, in the region of the groin, sometimes in other places, too, for observation of the vessels. After this preparation, the animals were subjected to the effect of an increased pressure of 6 psig for two hours and subsequent rapid (taking 3-3.5 min) decompression to the surface. Pressure values and exposure times were selected in preliminary experiments in such a manner that serious, life-threatening, or even fatal, decompression disturbances would develop in the animals.

As a result of the indicated effect, in all 18 dogs a violent development of aeroembolic phenomena took place, which, as the experiments showed, even determined by themselves the course and outcome of the decompression disorders. In exactly half of the cases, the matter terminated in the animals' death, seven dogs died 15-33 min and 2, 58 and 60 min after decompression, when there developed a total aeroembolism that was incompatable with life. The nine remaining dogs endured the critical period of decompression disorders. In this period, the aeroembolic phenomena in them also attained a very high intensity and assumed a threatening character, but then gradually weakened and passed away. These dogs were killed at different times (from two to four hours) after decompression.

У145

Such different outcomes of experiments involving one and the same external effect, independently of whether they can be explained by different deepnesses of narcosis or individual differences in the dogs, bear witness to the exceptionally great significance of purely physiological factors in the development

of decompression disturbances. Aeroembolic phenomena began in the dogs that died, as well as in those that survived, in the very first minutes (2-5 min) after decompression.

The first gas bubbles usually appeared in the fine veins coming from sections of tissue particularly rich in fat (veins of the inguinal fold, mammary gland and individual regions of the subcutaneous adipose cellular tissue). In a very short time, amounting to no more than a few tenths of a second, gas bubbles could be seen in the transparent fine veins, and soon they were already turning up in the greater part of the venous vessels situated in the subcutaneous cellular tissue as well as in the exposed sections of thorax and stomach (superficial muscular veins). The aeroembolic process had a very sudden beginning: no more than a few minutes after the appearance of the first single gas bubbles, intensive mass formation of gas developed throughout the visible peripheral vascular network. Replacing the bubbles continuously departing with the blood for the larger veins, from the periphery new ones arrived in an even greater number; side by side with the fine bubbles, ever larger ones appeared, attaining the diameter of the lumen of the vessels, and ever more voluminous, elongated accumulations of gas were formed. It became noticeable that in veins containing such gas plugs, the blood flow was beginning to slow down; at the same time, in a number of cases, gas bubbles were beginning to be formed in the corresponding arteries, too. But standstill of the blood flow had not yet set in even when there proved to be more gas in the fine veins than blood. It could be seen how the massive accumulations of gas overfilling the vessels and separated only by small bridges of blood were continuing to move slowly in the direction of the large veins and the heart.

Seeing what a great number of gas bubbles were continuously rushing into the large veins from those limited vascular regions that were accessible to our observations, we could easily imagine /146 what an abundance of gas was arriving in the right ventricle with the total venous blood. Involuntarily we asked ourselves: how can the blood circulation and the dogs' life be maintained under such conditions? To this question there can be only one answer: at the same time that gas was being formed in the venous blood, the air emboli entering the pulmonary vessels were undoubtedly being constantly liquidated. Had this not been so, then all of the ramifications of the pulmonary arterial network and, moreover, the cavities of the right side of the heart, too, would very quickly have turned out to be entirely filled with gas, and the blood circulation would immediately have come to a standstill. Experiments showed, however, that even dogs foredoomed to death could resist the violent development of aeroembolic phenomena for 15-30 min and even a whole hour, and half the animals proved able to overcome the noxious effect of aeroembolism.

Increasing gas formation in the peripheral vessels accessible to our observation (chiefly veins) ultimately led to circulatory

arrest in them. In the antemortem period of dogs that died from caisson phenomena, the blood circulation ceased in all peripheral vessels that we could observe. At the same time, in one experiment we detected a very curious fact: the retrograde movement of gas bubbles in a fine artery. This fact bears witness to the very high intensity of the process of gas formation in the peripheral ramifications of the vascular network. Apparently the nitrogen supersaturating the blood changes over from the dissolved state to the gaseous phase with so much energy that the pressure of the gas being formed exceeds the hemodynamic pressure in the fine artery in question.

In dogs that survived, gas formation in the vessels was generally somewhat weaker than in those that died, but attained a very high intensity, too. In the critical period of decompression disorders, in these animals, too, matters progressed to circulatory arrest in individual raminifactions of the peripheral vascular network. Subsequently, however, the aeroembolic phenomena subsided, the blood circulation that was disturbed in a number of vessels was restored and at the end of 1,5-2 hours after decompression, the blood proved to be completely free of gas bubbles. The changes in respiration and cardiovascular activity in all of the experimental dogs were very constant and were directly connected with the development of aeroembolic phenomena.

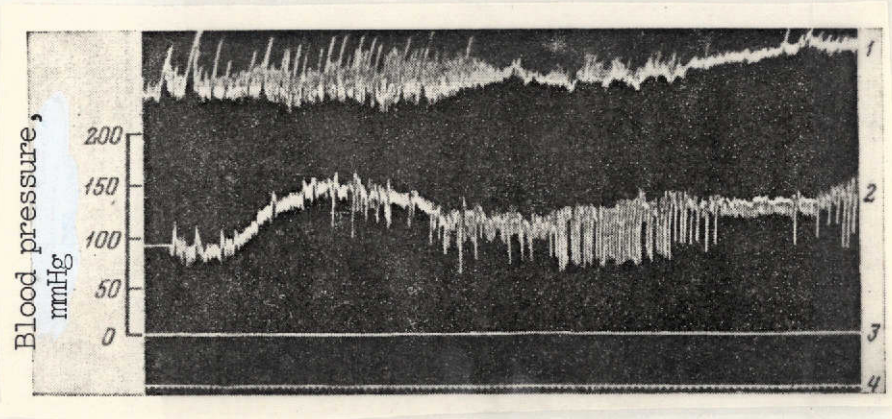

Fig. 35. Changes in respiration and blood pressure in dogs with serious decompression disturbances in 2-6th min after 2-hour exposure to a pressure of 6 atm.

1 -- respiration; 2 -- blood pressure; 3 -- zero line of blood pressure; 4 -- time indication, 3 sec.

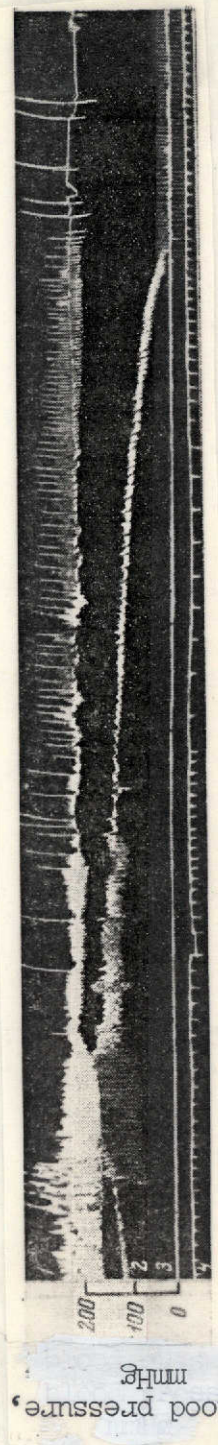

Fig. 36. Changes in respiration and blood pressure in 5-14th min after 2 hours exposure to a pressure of 6 atm in dogs with a fatal form of decompression disturbances. Time indication (4), 5 sec; in middle of recording, 16 sec. Rest of notation same as Fig. 35.

Simultaneously with the appearance of gas bubbles in the peripheral vessels, in all cases without exception panting arose, the pulse rate increased and the blood pressure rose. All these shifts, as well as the process of gas formation, had a sudden beginning and were very pronounced. The respiration rate increased to 100/min, and in some experiments, to 160/180 per min incident to a simultaneous considerable increase in the amplitude of respiratory movements. Very characteristic /148 was the emergence against this background of individual, sharp, even deeper inspirations, which is typical of acute cases of hypoxemia. The rise in the blood pressure ranged from 20 to 80 mmHg; in 1.4 of the cases, it just coincided with the onset of panting, in 1/4 of the cases, it somewhat preceded it and in half of the experiments, it took place with some delay. The rise in blood pressure always took place against a background of an increase in pulse rate; in 1/3 of the cases, this increase set in 30-60 sec earlier than the rise in blood pressure. The pulse rate increased by 30-60%, and in a number of experiments, it doubled its previous rate. In 1-3 min, when the rise in blood pressure was reaching its maximum, the increase in pulse rate against the background of ever intensifying panting was usually replaced by bradycardia. In individual cases, bradycardia began even earlier, during the rise in blood pressure.

The described reactions of respiration and the cardiovascular system, as has already been pointed out, took place in all dogs without exception; in

individual animals, these reactions were distinguished only by their degree of manifestation but were in all cases characterized by suddenness and abruptness (Fig. 35). Subsequently, the changes in blood pressure, respiration and pulse in different dogs differed as a function of the seriousness of the caisson phenomena and their outcome. In the most serious cases, which terminated in the rapid death of the animals (Fig. 36), against a background of violent panting the blood pressure after its first rise began to drop steadily and rather sharply and in 15-20 min reached critically low values. The bradycardia that had accompanied the pressor effect was replaced by an increase in pulse rate that progressed right up to the beginning of agonal disorders.

Panting continued in general right up to the death of the animal, its character changing in a very regular manner.

Periodic sharp and deep breaths kept arising ever more frequently, while superficial respiratory movements kept decreasing in amplitude. The relation between these and others changed: if at first respiratory movements that were more frequent and deeper than the usual constituded the main background against which separate sharp breaths arose, then the latter subsequently became predominant and followed one another at a certain ever accelerating pace. Fine respiratory movements gradually disappeared entirely, and only sharp deep breaths were left. This was followed by agonal inhibition of respiration, which was the background against which a few last, particularly sharp, respiratory movements arose at small and irregular intervals. Sometimes these antemortum respiratory movements arose in separate groups. This whole picture resembled extremely closely the changes in respiration incident to hypoxemia that progresses and terminates in death.

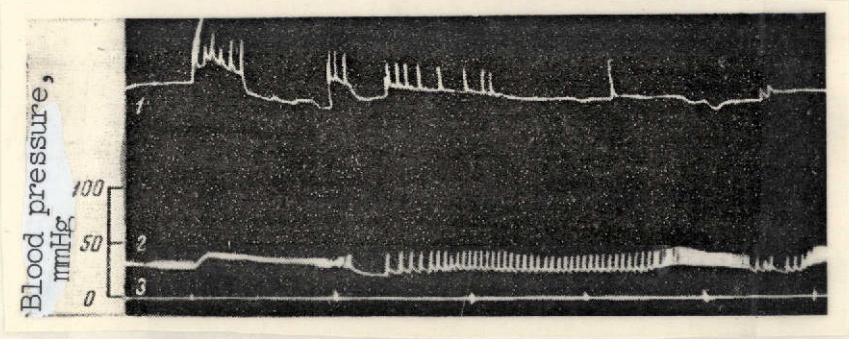

Fig. 37. Changes in respiration and blood pressure in dog in agonal period in 49-54th minute after decompression (6 atm, 2 hours).

1 -- respiration; 2 -- blood pressure; 3 -- time indication (1 min) and zero line of blood pressure.

A characteristic of rapidly developing fatal cases of /149
caisson disorders was abundant foaming from the tracheotomic tube incident to sharp panting. Sometimes the entire lumen of the tube became filled with foam that flew out with each exhalation. There can be no doubt that this indicated the development of edemata of the lungs. In animals that died from caisson phenomena a longer time after decompression, the reactions of respiration and of the cardiovascular system did not differ in character, but were correspondingly more stretched out in time.

Of interest are the changes in heart action that arose in dogs in the agonal period (Fig. 37). Against a background of periodic, attenuating respiration and extremely low blood pressure, the heart now increased its rate of beating, now slowed down or even stopped beating temporarily. The alternating periods of rapid and slow pulse were of different durations and did not show, as they do under other conditions, any clear connection with respiratory movements. It is safe to assume that these unusual changes in heart action were to a considerable extent due to an accumulation of gas in the heart and aeroembolic phenomena in the coronary vessels, leading to a disturbance of the supply to the heart muscle and conduction system elements.

Also noteworthy is the fact that heart action in the dogs that died from caisson phenomena was maintained longer than respiration, although the main assault incident to the development of aeroembolism, as is clear from all of the foregoing account, was directed at the blood circulation and at making difficulties for heart action. Here again we can see the similarity of critical decompression disorders to the phenomena of fatal hypoxemia.

In dogs that did not die from caisson phenomena, the /150
initial sudden changes in respiration and cardiovascular activity, which attained a maximum in the 30-40th minute after decompression, began gradually to smoothen out in conformity with the weakening of aeroembolic phenomena. The blood pressure at first dropped rather abruptly, and then extremely slowly and gradually, right up to the end of the experiment (killing of the animals). Its level dropped below the initial value some more during pronounced aeroembolic phenomena and, at the end of the experiment, fell to 40-50 mmHg. The bradycardia that had arisen in the period of most pronounced aeroembolic phenomena weakened and was replaced by an increase in pulse rate. The latter progressed regularly together with the drop in blood pressure, and, at the end of the experiments, the pulse rate sometimes amounted to 220/240 strokes per minute. Panting gradually smoothened out, too, and the deeper and more infrequent separate breaths became ever rarer and disappeared; 1.5-2 hours after decompression, when the gas bubbles were disappearing from the blood, uniform, shallow and somewhat accelerated breathing

was established. By the time the animals were killed, the state of the respiratory and circulatory functions presented no immediate danger to their life and was typical of every crucial experiment that lasted hours.

Autopsy of the animals that had died from caisson phenomena invariably revealed in the large veins and right side of the heart such an abundance of gas as absolutely precluded the possibility of blood circulation and was incompatible with life. The peripheral subcutaneous veins were everywhere overfilled with gas; great quantities of gas were detected in those veins of the peritonial and thoracic cavity that carry off the blood from fatty accumulations (perinephric adipose cellular tissue, omentum, fatty layers of the mediastinum). Incomparably fewer gas bubbles were in the muscular and mesenteral veins. Gas bubbles were also found in a number of arterial vessels; true, their appearance in the arterial blood, as observations showed, was to a considerable extent caused by postmortem gas formation. Gas bubbles were usually detected in the veins, as well as the arteries, of the coronary system immediately after opening the animals.

Extravascular accumulations of gas were also noted, in particular in the intermuscular interstices, in the joints and within the tubular bones, as well as gas bubbles in the adipose tissues. Very acute changes were detected in the lungs of dogs that had died of caisson phenomena. Large sections of the lungs, chiefly their dorsal sections, were dark red, consolidated and flabby and contained little air; when they were cut open, they excreted an effervescent bloody fluid -- in a word, all the signs of acute hemorrhagic edema of the pulmonary tissue were present. In other, less changed sections of the lungs, individual bluish-red foci and more or less densely disposed petechial hemorrhages were detected. /151

In dogs that had endured the critical period of decompression disorders and were killed at different times after decompression, autopsy revealed single gas bubbles in the peripheral veins, joints and adipose tissue only in individual cases. These cases pertain to experiments in which the animals were killed shortly after relative stabilization of the respiratory and circulatory indices (2-3 hours after decompression). In dogs that had been exposed for a long time, no gas bubbles were visible anywhere. Noted in the lungs of these animals, moreover, were clear local changes in the form of separate cyanotic blotches, as well as foci of fine hemorrhages and petechiae.

In a special series of experiments, with the aid of gas traps we studied decompression disorders of various degrees of manifestation. This second version of the experiments was con-

conducted on 39 dogs and ten cats.[11] As in the preceding series, the dogs were anesthetized with morphine and urethane, and the cats, with urethane alone. After anesthetization, both species were injected intravenously with 0.1 mg heparin per 1 kg. weight; the femoral (and, in a number of experiments, the cervical) vessels were prepared for recording the arterial pressure and introducing the gas traps. In experiments involving short-lived exposure of the animals to pressure, the gas traps were inserted before compression; in experiments involving prolonged exposure, they were inserted either before decompression, in which case the experimenter alone or with an assistant entered the chamber through the airlock, or immediately after decompression. We used pressures ranging from 1.2 to 13.5 psig; exposure varied from 10-15 min (for maximum pressures) to 5-6 h (for medium and low pressures).

Our first task was to determine the minimum depth after a stay at which gas bubbles begin to form in the blood, i.e., at which aeroembolic phenomena arise.

On five dogs and three cats, we carried out experiments with a 6-hour exposure of the animals to pressures of 1,6, 1,4 and 1.2 psig and subsequent rapid decompression to the surface. After exposure to a pressure of 1.6 psig (one experiment on a dog, and one on a cat), gas formation in the venous blood was quite distinct. Ten-twelve min after decompression, fine (about 1 mm in diameter) gas bubbles began to appear in the gas traps /152 inserted in the animals' femoral veins. In 15-20 min, the gas trap in the dogs accumulated about 20 such bubbles, and the gas trap in the cats, 6-8. Subsequently we could not make out their number. After exposure to a pressure of 1.4 psig (two experiments on dogs, one on a cat), the appearance of gas bubbles was noted in all three cases, too, but there were about half as many.

Two experiments on dogs and one on a cat involved a pressure of 1.2 psig. Twelve-fourteen min after decompression, in the gas traps inserted in the femoral veins of one of the dogs and the cat, single gas bubbles appeared that were even finer than those in the preceding experiment. We did not manage to detect them in the second dog.

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[11] Participants in a number of these experiments were A. A. Savich and K. S. Yurova, as well as Pintilie and Teodorescu -- our colleagues from Romania, who were taking advanced courses in the Department of the Physiology of Work of the S. M. Kirov Military Medical Academy.

These heuristic experiments showed that single gas bubbles can already form in the venous blood of the living organism after a stay at a depth of 12-14 m.

As has already been pointed out, the main task of the experiments using gas traps was to compare in the course of development of caisson phenomena the changes in respiration and cardiovascular activity with the intensity of gas formation in the blood. In experiments on 32 dogs and six cats, we traced decompression disorders that differed in seriousness, rapidity of development and outcome.

The experiments showed that the changes in respiration and cardiovascular activity incident to the development of decompression disorders are constantly and directly dependent on the intensity of gas formation in the venous blood.[12]

In a number of cases, the appearance of single fine gas bubbles in the gas trap introduced into the femoral vein was not accompanied by any changes in respiration, pulse or blood pressure. The reason for this is apparently either that single fine bubbles are a subliminal stimulus and generally cannot, when they are few in number, elicit responses from respiration or blood circulation or that these responses prove to be to some extent depressed by narcosis.

The first changes in respiration and cardiovascular activity could be observed in those cases where the gas bubbles in the gas trap appeared at once in great numbers (5-10 bubbles and more) or where their number increased more or less rapidly after appearance of the first single bubbles.

At the same time, an increase in rate, and in a number of cases, a deepening of respiration took place, as well as an acceleration in the rhythm of the heartbeat. If the decompression effect was not distinguished by high intensity and the aeroembolic/153 process did not progress, the matter was confined merely to inconsiderable panting and tachycardia.

Incident to the violent development of aeroembolic phenomena, when the number of gas bubbles in the gas trap increased so rapidly that it was soon impossible to count them, the reactions of respiration and of the cardiovascular system had an abrupt character, similar to that which was noted in the experiments of

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[12] In 20 experiments, gas traps were inserted not only into the veins, but also into the arteries of experimental dogs. The data on the possibility of gas formation in the arterial blood will be examined in the last section of the present chapter, which is specially devoted to this question.

the preceding series. Intensive panting developed, with periodically arising sharp, deep breaths; pulmonary ventilation, which we recorded on a kymograph in a number of experiments, increased by a factor of 4-6 and more, and the blood pressure rose against a background of sharp tachycardia. The quantity of gas in the gas trap in such cases increased rapidly, the upper part of the instrument kept filling with foam; the boundary line between foam and blood moved down before our eyes under the effect of new batches of gas entering the gas trap. The tachycardia in this case was soon replaced by a slowing down of the pulse, the blood pressure stopped rising and began to drop. If the aeroembolic process progressed further and the quantity of gas in the gas trap kept increasing, the blood pressure kept dropping steadily; at the same time the cardiac rate increased, the character of the panting changed as described above and the matter ended with the animal's death.

Incident to the development of the agonal stage of decompression disturbances, the gas trap turned out to be completely filled with gas and foam.

In those cases where decompression disorders did not lead to the dogs' death and functional shifts involving respiration and blood circulation abated and smoothened out, it was always possible to see how at the same time the ingress of gas in the gas trap decreased, and then ceased entirely. This direct connection between intensity of gas formation in the venous blood and changes in respiration and cardiovascular activity emerged clearly and invariably in the course of individual experiments, as well as in comparing the results of different experiments.

In order to arrive at some quantitative estimate of the intensity of gas formation in the venous blood, in a number of experiments we took note of what quantity of gas was being accumulated in the gas trap over a certain interval of time (for example, every 10 min)[13].

The experiments showed that in all cases where 1-2 mℓ gas accumulated in the gas trap introduced into the dog's femoral vein, the organism pretty much compensates for the aeroembolic /155
phenomena: the blood pressure either drops insignificantly or keeps to its initial level, and the matter is confined merely to moderate panting and tachycardia (Fig. 38). Incident to more intensive aeroembolic phenomena, when the quantity of gas trapped

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[13] Such determinations could not, of course, be completely accurate inasmuch as the gas in the gas trap is generally accumulated not in a pure form, but in the form of a more or less flocculent foam, and the boundary line between it and the blood is unclear.

in 10 min amounts to 4-5 mℓ, we note a drop in blood pressure by 20-40% from its initial value, considerably more intensive panting and a relative slowing down of the heartbeat (Fig. 39). But if the aeroembolic process progresses no further, the blood pressure becomes stabilized at the lower level and no life-threatening functional disorders set in. Under these conditions, too, compensation for aeroembolic phenomena is rather stable, though incomplete. And in a little while, the respiratory and circulatory functions become normalized.

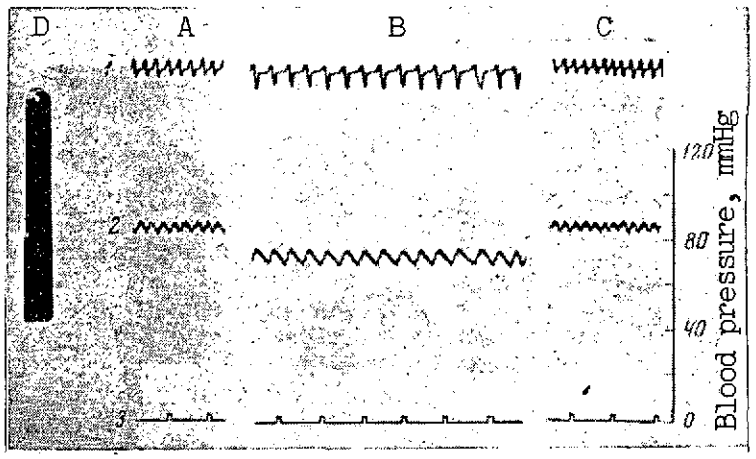

Fig. 38. Changes in respiration and blood pressure in anesthetized dog incident to aeroembolism arising after single-step decompression from under a pressure of 6 psig (1-hour exposure). Quantity of gas accumulating in gas trap in 10 min ~ 5-2 mℓ (D); 1 -- respiration; 2 -- blood pressure in left femoral artery; 3 -- time indication (10 sec) and zero line of blood pressure; A -- before compression; B -- 12th minute; C -- 60th minute after decompression.

Critical disorders of respiration and blood circulation arise only when the quantity of gas entering the gas trap is still greater and reaches 10-15 mℓ and more in 10 min (Fig. 40). In these cases, too, the animals died not at once; even with such intensive gas formation, their life was maintained for 15-20 min. /156

In evaluating the above facts, it must be taken into account that in the experiments, the quantity of gas formed in only one of the femoral veins was determined. The volume of blood passing through the anesthetized dog's extremity amounts to only a small fraction of the total volume of blood circulating in the organism. Consequently, the total quantity of gas making its

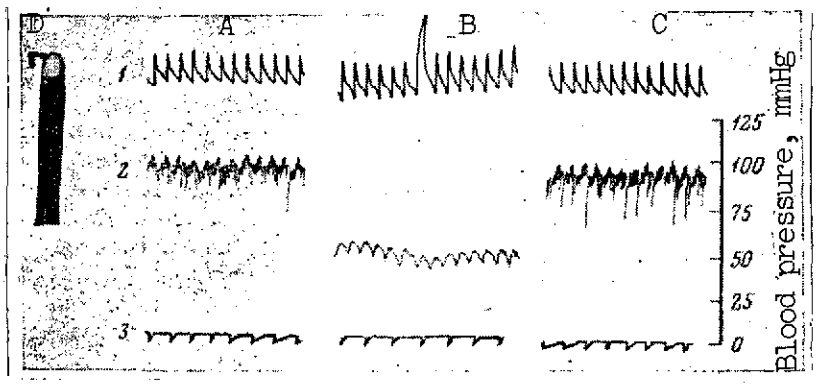

Fig. 39. Changes in respiration and blood pressure in anesthetized dog incident to aeroembolism arising after single-step decompression from under a pressure of 6 psig (exposure: 1 h, 20 min). Quantity of blood accumulating in gas trap in 10 min ~ 5 min (D). Same notation as Fig. 38. Time indication, 5 sec. A -- before decompression; B -- 13th minute; C -- 60th minute after decompression.

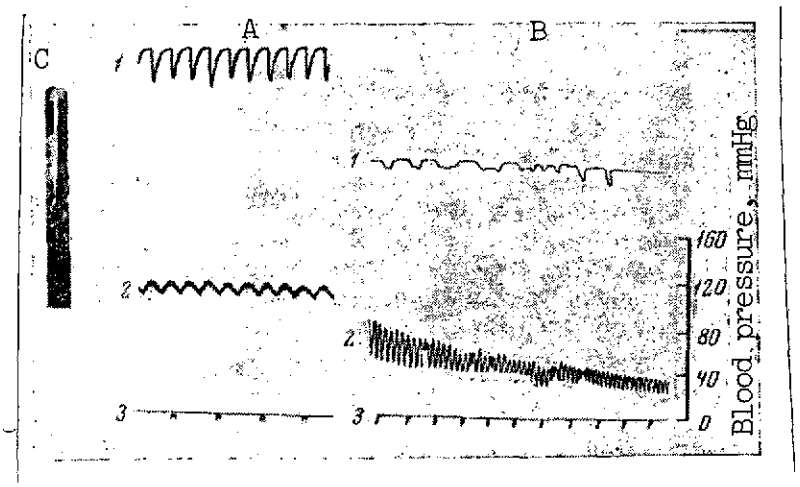

Fig. 40. Changes in respiration and blood pressure in anesthetized dog incident to fatal aeroembolism arising after single-step decompression from under a pressure of 6 psig (2-hour exposure). Quantity of gas accumulating in gas trap in 10 min ~ 13-14 mℓ (C). Same notation as Fig. 38. Time indication, 10 sec. A -- before decompression; B -- 15th minute after decompression.

way with the venous blood to the right side of the heart and, further, to the pulmonary arteries exceeds by far the above figures.

Thus, the experiments with gas traps confirmed the results of experiments with visual observation of gas formation in vessels. Firstly, they showed the direct connection between intensity of gas formation in the venous blood and changes in respiration and cardiovascular activity in the process of development of decompression disorders and, secondly, brought out the organism's exceptionally powerful compensatory resources, which permit it to cope with aeroembolic phenomena even when their intensity is very high.

Artificial Aeroembolism

Inasmuch as crucial experiments showed that the functional shifts that are characteristic of decompression disturbances depend directly on the intensity of gas formation in the venous blood and the quantity of gas going with the venous blood to the right side of the heart and to the lungs, it was natural to undertake experiments involving the injection of air into the animal's veins and to compare the consequences of such artificially induced aeroembolism with the picture of decompression disorders. Having already convinced ourselves in the first experiments of the great similarity of functional shifts in the two cases, we did not confine ourselves to comparison of decompression aeroembolism with aeroembolism induced by injecting air, but also investigated what effect is exerted on the organism by intravenous administrations of helium, oxygen and carbon dioxide. The study of the organism's reactions to intravenous administration of different gases was of interest in connection with the fact that gas bubbles incident to real caisson phenomena can differ considerably in composition, depending, firstly, on what gaseous mixture the man breathed before decompression, and, secondly, on the absolute values of the pressure at which decompression disorders developed in him. The lower this pressure, the greater the ease with which carbon dioxide and oxygen molecules will diffuse into the gas bubbles formed mainly on account of an indifferent gas (nitrogen or helium). In this connection, it was of interest to study the effect of each of the above-mentioned gases separately, as well as of air and the helium-oxygen mixture that are used in diving practice.

On six dogs, 50 cats and 18 rabbits, we conducted 74 /157
crucial experiments using urethane (in the case of the dogs, morphine-urethane) narcosis and recording on a kymograph the arterial blood pressure and respiration, and 15 experiments on unanesthetized rabbits involving observation of the animals' general condition after intravenous administration of the gases. The blood pressure in the crucial experiments was recorded in

the femoral artery, and the gases were administered into the femoral vein of the contralateral side with the aid of the above-described system.

In all, we administered 110 injections of air, 26 of a helium-oxygen mixture, 40 of oxygen and 15 of carbon dioxide. The doses, rate of administration and sequence of using the gases in individual experiments varied as a function of the purpose of the experiment.

The experiments[14] showed that the reactions of respiration and blood circulation to intravenous administrations of air were very close to those that occur incident to decompression aeroembolism. Here, too, the net result is rapidly increasing panting with the characteristic, periodic, deep, sharp breaths of incipient tachycardia, which was soon replaced by a slowing down of the pulse and a drop in blood pressure. In contrast to the reactions that take place incident to postdecompression aeroembolism, intravenous administration of air, even when effected at a high rate, is usually not accompanied by an initial rise in blood pressure. It must be assumed that this short-lived pressor effect that generally accompanies a violent onset of decompression aeroembolic phenomena is connected with the formation of gas bubbles at the periphery of the greater circulatory system. It is probable that the gas bubbles formed in the fine vessels either by themselves or by means of a responding angiospasm increase peripheral resistance, which leads to an initial rise in arterial pressure.

The ingress of gas into the vessels of the lesser circulatory system, which leads to a block of the pulmonary capillaries and a drop in arterial pressure, in this initial period does not yet reach its maximum and does not prevent the development of the pressor effect. But this moment soon acquires a determinative significance, as the pressor effect is replaced by a progressive drop in blood pressure. It goes without saying that incident to the intravenous introduction of air, we cannot reproduce the aeroembolic phenomena that take place at the periphery of the vascular system after decompression but can do no more than imitate the net result of the aeroembolic process: the ingress of gas bubbles with the total venous blood into the right side of the heart and, further, into the pulmonary vessels. The fact that intravenous administration of air very fully reproduces the /158 general picture of the functional shifts incident to development of caisson phenomena points to the decisive role of aeroembolism of the pulmonary vessels in the pathogenesis of decompression disorders.

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[14] Experiments conducted jointly with K. S. Yurova.

The great similarity of the changes in respiration and cardiovascular activity incident to artificial and decompression aeroembolism appears clearly also throughout the subsequent course of the experiments. In those cases where moderate doses of air are used and the rate of its administration is kept within certain limits, the functional shifts, just as in the case of nonfatal decompression aeroembolism, after attaining a maximum, begin gradually to smoothen out at the end of the injection and, after a certain time, disappear. When increased doses of air are injected more rapidly, the matter ends in the organism's death attended by the same phenomena that occur in fatal cases of decompression disorders. Panting in this case continues right up to agony, but changes its character in a typical manner: the amplitude of the finer respiratory movements constituting the main background of the pneumogram gradually grow shorter, and sharp deep breaths arise ever more frequently and rhythmically. The blood pressure drops steadily, and as it does so, the pulse rate progressively increases. Death sets in from respiratory standstill, which passes through a stage of periodic breaths of the Cheyne-Stokes type; the heart outlasts respiration for a longer or shorter time, displaying in a number of cases unexpected transitions now to a slower, now to a faster rhythm.

Comparison of Figs. 41 and 42, which are kymograms of the changes in respiration and cardiovascular activity (experiments on cats) incident to decompression and artificial aeroembolism, shows that these changes, if we except the short-lived initial pressor effect that is characteristic of decompression aeroembolism, are very much alike.

It should be noted that this similarity may not be achieved, if the intravenous air injection is effected too rapidly. In this case there is always the danger (especially in experiments on cats and rabbits) of causing a gas block of the right atrium and the animal's sudden death from phenomena that are characteristic of neither decompression aeroembolism or gradually increasing artificial aeroembolism.

Experiments with intravenous injection of different gases showed that they differ sharply in the intensity of their effect. It turned out that the organism endures an intravenous injection of nitrogen and air worst of all, helium and a helium-oxygen mixture considerably better, oxygen still better and, finally, carbon dioxide especially well. These relationships are illustrated by the kymograms of Figs. 43, 44 and 45. The effects shown in the kymograms refer to cats, which, as the experiments showed, are incomparably more resistant to intravenous gas injection than rabbits. Thus, injection of 1-1.5 ml of air every 15 sec very often proves to be fatal for rabbits, while cats endure injection of 10-15 ml of air at the same rate. /160

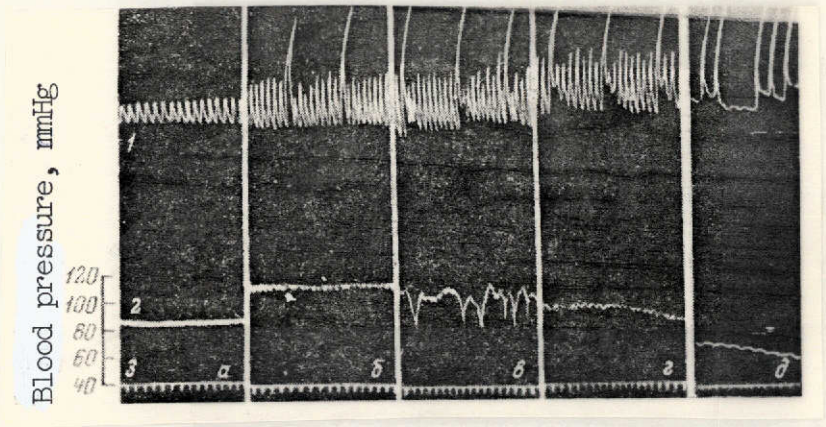

Fig. 41. Changes in respiration and cardiovascular activity incident to decompression aeroembolism after exposure to a pressure of 7 atm for two hours. A -- before decompression; B -- 3rd; C -- 5th; D -- 9th; E -- 16th minute after decompression for 1 h, 20 min; 1 -- respiration; 2 -- arterial blood pressure; 3 -- time indication, 2 sec.

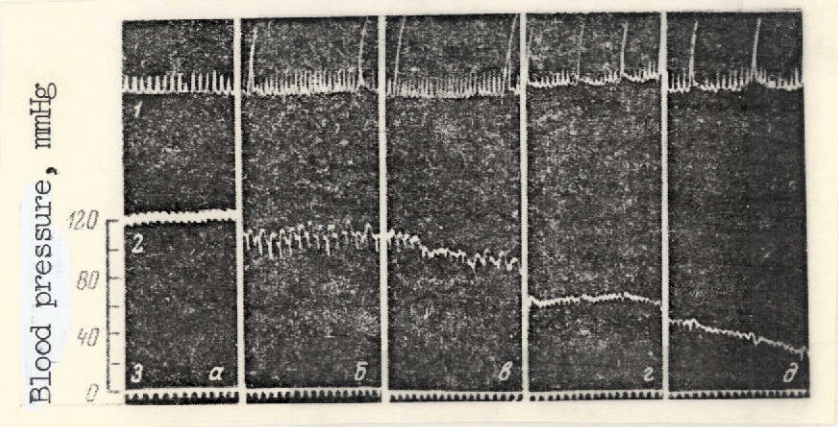

Fig. 42. Changes in respiration and cardiovascular activity incident to air injection into femoral artery at a rate of 2 mℓ/min. A -- initial background; B -- 2nd; C -- 4th; D -- 7th; E -- 20th minute after start of injection. Same notation as in Fig. 41.

Fig. 43 shows the effect of injecting 14 mℓ of air into the femoral vein of a cat at a rate of 1 mℓ every 14 sec. After a short-lived slowing down of respiration, panting arose and the blood pressure dropped from 110 to 24 mmHg. At the end of the air injection, a gradual restoration of functions was noted,

but even 27 min after the injection (the last recording on the right), the blood pressure had not yet returned to its initial value.

Fig. 44 shows the effect of injecting the same amount of helium at a somewhat faster rate (1 mℓ every 12 sec). As the kymogram shows, the changes in respiration and cardiovascular activity were less considerable: the blood pressure dropped from 112 to 40 mmHg, but as early as 3 min after the injection, it almost attained its original level.

Fig. 45 shows the effect of oxygen injected into the vein at a rate of 1 mℓ every 13 sec. The initial sharp drop in the blood pressure is explained by the fact that at this moment, the rate of administering the gas was increased. Subsequently, however, as the kymogram shows, incident to injection of 14 mℓ O_2, the blood pressure remained practically unchanged, and panting was not very pronounced. Critical functional disturbances arose only after injection of 41 mℓ O_2.

Experiments with intravenous injection of carbon dioxide at the same rate (Fig. 46) showed that the animals can counteract the given effect for an indefinitely long time. In this case the blood pressure does not change appreciably and only steady and intensive panting arises. As Fig. 47 shows, even jerky intravenous administration of very great amounts of CO_2 (32 mℓ every 12 sec) causes only a short-lived drop in blood pressure and very fleeting changes in respiration: functions are restored in a very few minutes.

The considerable differences in the effect of different gases incident to their intravenous administration are apparently connected with two circumstances. The first concerns chemically active gases, namely O_2 and CO_2, and resides in the fact that upon being injected into the blood, they can to one extent or another combine with substances that the blood contains (O_2 with hemoglobin, and CO_2 with alkalis).

The second circumstance refers to all the investigated gases and has to do with their different diffusibilities. As is well-known, carbon dioxide is much more diffusible than oxygen, and helium, much more so than nitrogen. For the last two gases, which are chemically inert, the difference in their diffusibilities is the decisive and only cause of the difference in intensity of their physiological effect.

The course and outcome of artificial aeroembolism will depend, in the first place, on the rate of liquidation of the gas bubbles arriving in the pulmonary capillaries and blocking them. And their liquidation will be determined by the rate of diffusion of the gas from these bubbles into the alveolar space. У161

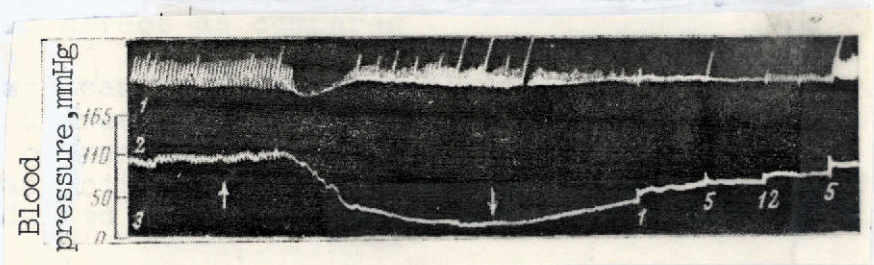

Fig. 43. Changes in respiration and blood pressure in anesthetized cat incident to intravenous injection of 14 mℓ air at a rate of 1 mℓ every 14 sec. 1 -- respiration; 2 -- blood pressure; 3 -- time indication (1 sec) and zero line of blood pressure. The arrows point to the beginning and end of the air injection; the figures below the pressure curve are interruptions in the recording (in min).

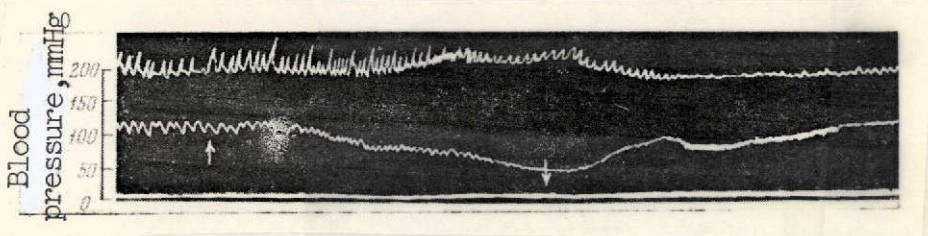

Fig. 44. Changes in respiration and blood pressure in anesthetized cat incident to intravenous injection of 14 mℓ helium at a rate of 1 mℓ every 12 sec. Same notation as in Fig. 43.

The faster liquidation of helium emboli owing to the greater diffusibility of helium serves to explain also its milder physiological effect as compared with nitrogen.

This fact can to a certain extent explain the apparent discrepancy between the data of A. P. Brestkin [1958] on the coefficient of maximum permissible supersaturation of solutions for helium and nitrogen and the results of the investigations that he conducted jointly with I. A. Aleksandrov [1964] of the coefficient of safe supersaturation of the human organism with these gases. It was demonstrated in A. P. Brestkin's experiments with solutions in vitro that helium forms bubbles in solution

at lesser degrees of supersaturation than does nitrogen; but it was established in the investigation of A. P. Brestkin and I. A. Aleksandrov that the development of caisson symptoms in human beings emerging from under increased pressure takes place /162
incident to breathing a helium-oxygen mixture at a greater supersaturation than in the case of breathing air.

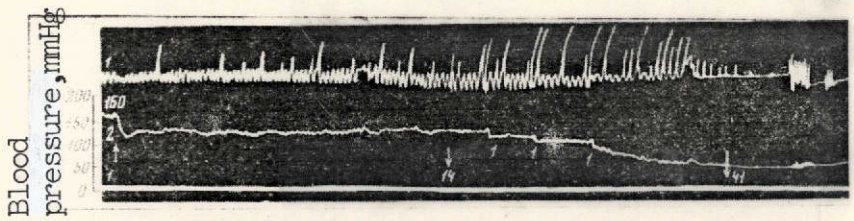

Fig. 45. Changes in respiration and blood pressure in anesthetized cat incident to intravenous injection of oxygen at the rate of 1 mℓ every 13 sec. The arrows point to the beginning and end of injection of 14 and 41 mℓ O_2. The notation is the same as in Fig. 43. The figures under the blood pressure curve are interruptions in the recording (in min).

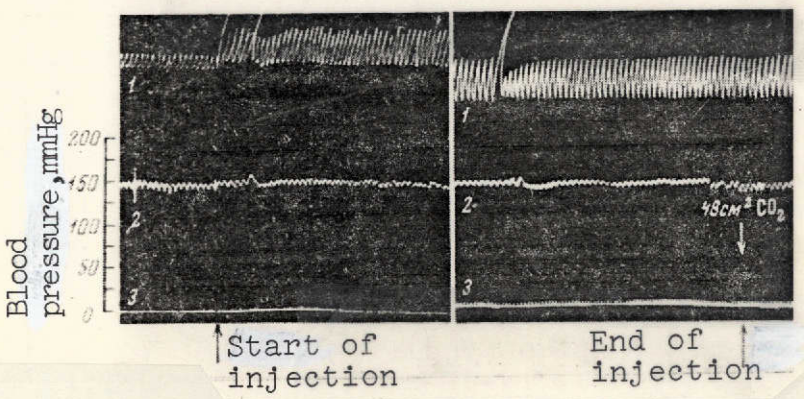

Fig. 46. Changes in respiration and blood pressure in anesthetized cat incident to intravenous injection of 48 cm^3 carbon dioxide at a rate of 1 cm^3 every 10 sec. Same notation as in Fig. 43.

It is probable that incident to the organism's supersaturation with helium, gas bubbles, just as in experiments in vitro, are more readily formed than incident to supersaturation with

nitrogen, but owing to the greater stability of nitrogen bubbles, decompression symptoms incident to breathing air develop at lesser degrees of supersaturation.

In the course of the experiments involving injection of different gases, still another circumstance of theoretical, as well as practical, interest was brought to light, namely: the greater or lesser ease with which the organism endures an injection of one gas or another depends to a considerable extent on the diffusion relationships created in the lungs between the air emboli and the alveolar gaseous medium. Thus, if the animal is /163 compelled to breathe oxygen, an intravenous injection of oxygen is endured less easily than under ordinary conditions. An injection of air incident to the breathing of oxygen, on the other hand, is endured considerably more easily. Similarly, an intravenous injection of helium is endured less easily in the event that the animal is breathing a helium-oxygen mixture, and more easily when air and, all the more, oxygen is breathed.

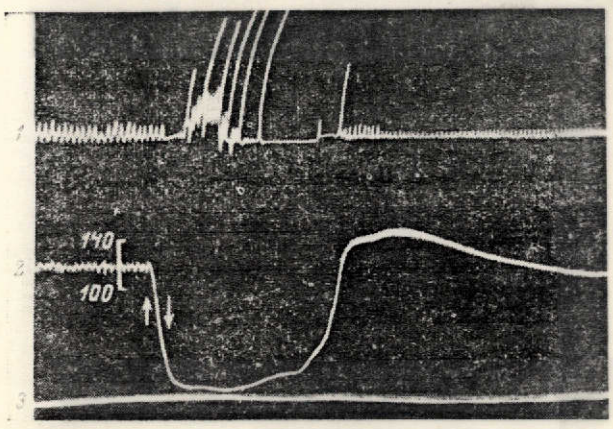

Fig. 47. Changes in respiration and blood pressure in anesthetized cat incident to intravenous injection of 32 mℓ CO_2 every 12 sec. The arrows point to the start and end of CO_2 injection. Same notation as in Fig. 43.

These facts confirm the conclusion that the gas bubbles arriving at the pulmonary capillaries with the blood are liquidated because the gas they consist of diffuses into the alveolar space. It stands to reason that the liquidation of air emboli in this way should take place more rapidly in those cases where their gaseous composition differs from that of the gaseous medium filling the alveoli.

The reservation must be made that the above-mentioned diffusion relationships, even though they change the effect of intravenous gas injection, do not by themselves determine those sharp differences in their physiological effect that were detected in the experiments. An air injection incident to the breathing of air is endured incomparably less easily than an injection of a helium-oxygen mixture incident to the breathing of such a mixture and, especially, than an oxygen injection incident to the breathing of oxygen. Consequently, the conclusion drawn above that the different

effects of gases incident to intravenous injection is determined by their nature and properties (for nitrogen and helium, this is, above all, their diffusibility), remains completely valid.

Finally, it should be noted that in the critical period of /164 aeroembolism, when the changes in respiration and blood circulation assume a threatening character, the breathing of oxygen proves to be useful, regardless of what gas is being injected into the vein. Its positive effect under such conditions apparently reduces to a lessening of the oxygen want caused by disorders of the hemodynamics and by impaired aeration of the blood in the lungs.

The experiments showed that, in spite of the different intensities of the effect of intravenously injected gases, the character of the changes in respiration and cardiovascular activity remains the same. In a class by itself is only carbon dioxide, incident to whose injection the reactions of the respiration and of the cardiovascular system show peculiarities and differ from the corresponding reactions incident to the injection of other gases. This is quite understandable inasmuch as carbon dioxide acts not only like a gas embolizing the pulmonary capillaries, but also like a specific stimulus of the respiratory and cardiovascular center. But as far as oxygen, nitrogen and helium are concerned, incident to their injection, as can be seen from the kymograms presented above, the changes in respiration and cardiovascular activity are monotypic in character. This, too, is quite understandable: the nature of the gas plugging up the pulmonary capillaries is of no great significance for those direct consequences that this plugging up induces; disorders of the hemodynamics and impaired arterialization of the blood will be determined by the number of blocked pulmonary capillaries, regardless of the composition of the air embolism with which their lumen is plugged up.

Summing up the net result of the experiments with intravenous gas injections, we can conclude that they have shown the great similarity of the functional shifts incident to artificial and decompression aeroembolism. In other words, they directly confirm the conclusion that aeroembolism of the venous system and pulmonary capillaries is the central phenomenon in the pathogenesis of decompression disturbances. These experiments have also confirmed the conclusion that the organism possesses exceptionally powerful compensatory resources in respect of the noxious effect of aeroembolism. Not to mention that the organism copes with very considerable doses of intravenously injected gases incident to individual injections, it proves to be able to endure repeated injections, when the total quantity of the gas exceeds, as it were, all permissible limits. Thus, in a number of 2-3-hour experiments, cats received 200-300 mℓ each of inert gases, and in experiments with oxygen, the total quantity of intravenously injected gases amounted to 500-600 mℓ, i.e., approximately twice

as much as the capacity of the vascular system, and nevertheless the animals, upon recovering from the next injection, showed no life-threatening functional disorders. These facts, as well as those cases of survival of animals incident to the development of serious caisson phenomena which were described in the previous /165 section, directly indicate that incident to the development of aeroembolic phenomena, a constant and very thorough liquidation of the gas bubbles appearing in the venous blood takes place.

On the permeability of the pulmonary capillaries to air emboli and on the possibility of postdecompression gas formation in the arterial blood

In connection with the conclusion about the role of aeroembolism in the development of decompression disorders, two questions arise: in the first place, does some part of the gas formed in the veins pass through the pulmonary capillaries into the arterial system and, in the second place, are air emboli formed in the arterial blood itself? If aeroembolic phenomena do take place in the arterial system, another question arises: what is their role in the development of decompression disorders? Sometimes enunciated in the practice of teaching and in some training manuals are groundless and categorical opinions of this last point, viz.: aeroembolism of the ramifications of the arterial network and, above all, the cerebral arteries is the cause of the entire symptom complex of serious decompression disturbances. Attempts have also been made to substantiate theoretically the great role of arterial aeroembolism in the pathogenesis of decompression disorders. Such a substantiation has been adduced, in particular, by M. I. Yakobson in developing his concept of the penetration of gas "seeds" -- centers of subsequent gas-bubble formation -- into the arterial blood from the lungs.

Before examining in greater detail the question of arterial aeroembolism, it should be noted that the idea of its leading role in decompression disturbances is already contradicted by the general picture of their development. This picture, as is already clear from the data presented above, is characterized by the monotypicity of the changes in respiration and blood circulation. In individual cases, the changes in these functions differ only in their greater or lesser degree of manifestation, but not in their character; moreover, they arrive in all cases not suddenly and abruptly, but grow more or less gradually. None of this fits into the picture of embolism of the cerebral arteries, incident to which the functional shifts may, depending on the localization of the emboli, be very diverse and, in critical cases, generally are sudden, rapid and irreversible. This is how matters stand incident to experimental embolism of the cerebral vessels by means of solid particles, especially a suspension of club moss; in pathological physiology, this method of procedure has

been elevated to the rank of lecture demonstrations that are infallible in their effect. The consequences of aeroembolism of the cerebral arteries are the same.

In terminating some crucial experiments, we injected air into the carotid arteries of the animals and, in conformity with what was said above, we detected preagonal and agonal changes /166 in respiration, blood pressure and heart action that were very diverse in character; in individual cases, the changes in question arose incident to very diverse quantities of injected air and did not directly depend on its dose, as it does incident to intravenous injections. It already follows from these facts that the described constant shifts in respiration and blood circulation incident to development of decompression disturbances cannot be explained by a plugging up of the cerebral arteries with air emboli.

To elucidate the question of the permeability of the pulmonary capillaries to air emboli (the data on this question in the literature, as was pointed out above, are contradictory), we undertook special experiments in which air was injected into the venous blood and checked for its appearance in the arterial blood with the aid of gas traps. In 12 experiments on dogs and nine experiments on cats, air was introduced into the femoral vein, and the gas trap was inserted in the carotid artery. The doses of air injected into the vein in each experiment was increased from time to time right up to the animal's death from one of the next injections; the rate of injection was varied in different ways. In no case were gas bubbles that were visible to the naked eye detected in the gas trap inserted in the carotid artery.

In four experiments conducted on cats, we changed the animals over to artificial respiration, opened their thorax and inserted a cannula in the right or left pulmonary artery for air injection and a gas trap in the corresponding pulmonary veins, right after which we began to perfuse the vessels of the given lung with physiologic solution. Then we injected air through the lateral outlet of the cannula inserted in the pulmonary artery and gradually increased the head of the perfusate and, consequently, the pressure under which the air was injected, too.

In no cases, not even when the pressure under which the perfusate and air entered the pulmonary arterial vessels exceeded the physiological limits and reached 200 and 250 mmHg, did we detect in the gas trap gas bubbles that were visible to the naked eye. Thus, the experiments led to the firm conviction that the pulmonary capillaries do not let pass gas bubbles that are visible to the naked eye. This, of course, does not preclude the penetration through the pulmonary capillary network of gas bubbles of microscopic size that are inaccessible to observation but does compel complete rejection of the hypothesis that air

emboli formed in the venous blood can, by passing through the pulmonary capillaries, develop their pathogenic effect in the arterial system, plugging up its ramifications.

The results of the described experiments naturally raised the question whether it is the pulmonary capillaries alone that possess this property of not letting pass freely the gas bubbles arriving in them, or whether this property is inherent in the capillaries of the greater circulatory system, too. In this connection, in experiments on cats, we injected air into different /167
arteries of the greater circulatory system and checked (visually or by means of gas traps) for its appearance in the corresponding veins. We effected 12 air injections into the femoral arteries, eight into the renal arteries, six into the splenic arteries, 11 into the mesenteral arteries and eight into the carotid arteries.

In all cases without exception, the air injected into the enumerated arteries immediately appeared in the corresponding veins. We could judge of the quantity of air passing into the veins only approximately, but there can be no doubt that it was close to the quantity of air injected into the artery. Thus, these experiments showed that the ramifications of the vessels of the greater circulatory system, in contrast to the pulmonary vessels, are permeable to air emboli. In considering these facts, the question inevitably arises whether the emboli pass only through the capillaries or through the arteriovenous anastomoses, too. The presence of such anastomoses is acknowledged by the majority of researchers for the ramifications of the greater, as well as the lesser, circulatory system. The results of our experiments give no grounds for disputing this but do show in all clarity that the ramifications of the pulmonary vessels and those of the vessels of the greater circulatory system deal differently with the air emboli entering them. The former hold them back reliably, the latter let them pass easily.

Leaving aside the problem of decompression disturbances, these facts ought to be viewed from the point of view of general and evolutionary physiology. It is possible that the impermeability of the pulmonary vessels to air emboli (and, we must imagine, to solid emboli, too) is a useful evolutionary acquisition. Under natural conditions of the organism's existence, emboli (of whatever origin) generally have as their point of departure the peripheral vessels of the greater circulatory system. Injuries to the periphery of the body are the most frequent traumas that animals encounter in the course of evolution. Incident to these traumas, the possibility of an embolus always arises -- from the penetration of fat particles into the vascular system (incident to bone fractures or massive injuries to the subcutaneous tissue); subsequently, incident to the development of the inflammatory process -- from the getting into the blood

of pieces of necrotized tissue, microbial accumulations, etc. The moxious consequences that can ensue from such emboli getting into the arterial blood are quite obvious.

In this connection, the thought involuntarily arises that the pulmonary vessels in the course of evolution acquired the role of a barrier that is impermeable to emboli, preventing them from getting into the arterial vascular system. In this connection, it must be remembered that emboli of one sort or another that are formed at the periphery have also another path -- through the lymph system. But on this path, even before they can get into the total venous blood, they encounter additional barriers -- the lymph nodes with their reticular trabeculae that filter out, /168 if one may say so, all extraneous particles reaching them.

Thus, to the first question that was asked above -- whether gas bubbles that are formed in the veins cannot penetrate into the arterial blood -- we can give a quite definite answer on the basis of experiments that have been carried out: the pulmonary vessels do not let pass into the arteries gas bubbles that are visible to the naked eye; in other words, they do not let pass air emboli that can cause a plugging up of the arterial vessels. But we still cannot conclude from these data that aeroembolic phenomena in the arterial system do not play an important role in the development of decompression disturbances. The second question remains -- are not air emboli formed in the arterial blood itself?

To investigate this possibility, in 20 crucial experiments on dogs we simultaneously recorded the appearance of gas bubbles in the venous and the arterial systems with the aid of gas traps after exposing the animals to different pressures and subsequently decompressing them to the surface.

In the first five experiments, we used a pressure of 3 psig with a 6-hour exposure. In all cases, 5±8 min after decompression gas bubbles began to appear in the venous gas trap; their number increased so rapidly that it was soon no longer possible to count them -- the entire upper part of the gas trap filled up with a layer of foam. Gas formation intensified in the course of the first 15-20 min; beginning with the 30-36th minute, it gradually decreased and 1-1.5 hours later, it had almost completely faded away. In three out of five experiments, in the 10-12th minute after decompression, we observed small numbers (10-12) of gas bubbles in the arterial gas trap; subsequently their number did not increase. None of the five animals died of caisson phenomena. In the remaining 15 experiments, we used pressures ranging from 5 to 13.5 psig with different exposures. The results of these experiments are shown in Table 7.

As can be seen from the table, in five of the experiments no bubbles were detected in the arterial gas trap. Intensive

TABLE 7. RESULTS OF EXPERIMENTS ON DOGS WITH ARTERIAL AND VENOUS GAS TRAPS

Weight of dog, kg	Pressure, psig; Exposure, min	Time of appearance of bubbles in gas trap (after termination of decompression), min						Outcome of experiment
		In arterial trap			In venous trap			
		Start	Maximum	End	Start	Maximum	End	
20	5.0(20); 4.0(120); 3.0(90); 4.0(90)	13	20-22	30-35	5	8-12	Gas formation continues until end of experiment	Death by asphyxiation 2 h 20 min after decompression
22	5.0(300); 4.0(28); 5.0(60)	Enormous number of bubbles immediately after opening of trap						Death in 4th minute from decompression phenomena
22	5.0(140); 3.0(30); 4.0(20)	7	10	10	4	15	Quantity of gas steadily increases	Death in 27th minute from decompression phenomena
10.3 (puppy)	7.0(30)	No gas formation			Immediately a great amount of bubbles		In 50th minute	Death by asphyxiation 2 h 30 min after decompression
22	8.0(35); 3.0(30); 6.0(80)	3	7-12	20	8	Gas formation progresses until end of experiment		Death in 45th minute from decompression phenomena
27	9.0(10)	No gas formation			10	Gas formation progresses until end of experiment		Death in 25th minute from decompression phenomena
25	9.0(10)	No gas formation		5	40	Continues until end of experiment		Death by asphyxiation 2 h after decompression

(TABLE 7 continued)

Weight of dog, kg	Pressure, psig; Exposure, min.	Time of appearance of bubbles in gas trap (after termination of decompression), min						Outcome of experiment
		In arterial trap			In venous trap			
		Start	Maximum	End	Start	Maximum	End	
20	9.0(20)	4	Uniformly intensive gas formation until animal's death		Immediately	Uniformly intensive gas formation until death of animal		Death in 18th minute from decompression phenomena
18,9	9.0(20)	4	5	19-20	Immediately	40-45	Continues until end of experiment	Death by asphyxiation 2 h 30 min after decompression
24	9.0(30)	Immediately	7-8	Continues until end of experiment	Immediately	Gas formation progresses until end of experiment		Death in 13th minute from decompression phenomena
18,5	9.0(30)	4	7-8	12	Injected in 10th minute after decompression. Abundant gas formation until end of experiment			Death in 13th minute from decompression phenomena
10	13.5(8)	No gas formation			-			Death by asphyxiation 3 h after decompression

(TABLE 7 continued)

Weight of dog, kg	Pressure, psig; Exposure, min.	Time of appearance of bubbles in gas trap (after termination of decompression), min						Outcome of experiment
		In arterial trap			In venous trap			
		Start	Maximum	End	Start	Maximum	End	
23.3	13.5(15)	Immediately	4-5	Continues until end of experiment	No gas trap introduced; great number of bubbles in veins			Death in 14th minute from decompression phenomena
23	13.5	Immediately	Gas formation progresses until animal's death		No gas trap inserted; great number of bubbles in veins			Death in 7th minute from decompression phenomena
20.5	13.5(15)	No gas formation			7	50	Continues until end of experiment	Death by asphyxiation 2 h 20 min after decompression

gas formation was noted in the venous blood of these five animals; one dog died of caisson phenomena.

In the remaining ten experiments, side by side with violent gas formation in the veins, gas bubbles appeared also in the arterial gas traps. In some cases, they were single, but in most cases, there were many dozens of them. In those experiments where rapid death of the animals did not set in, new gas bubbles ceased to be formed in the arterial gas traps after some time (most frequently after 10-12 min), whereas in the venous blood, violent gas formation still continued. Eight experiments terminated in the animals' death from decompression phenomena, six of the dogs dying in a relatively short time after decompression (from 4-18 min) and two of them, after a longer time (27 and 45 min). Particularly noteworthy is the fact that two dogs, in spite of extremely violent gas formation accompanied by the appearance of a great number of gas bubbles in the arterial gas trap, did not die from decompression phenomena; they died of asphyxiation 2 h 20 min and 2 h 30 min after decompression, when the aeroembolic process had almost completely faded away, the vessels were free from gas bubbles and the state of respiration and blood circulation presented no danger to the organism's life. /171

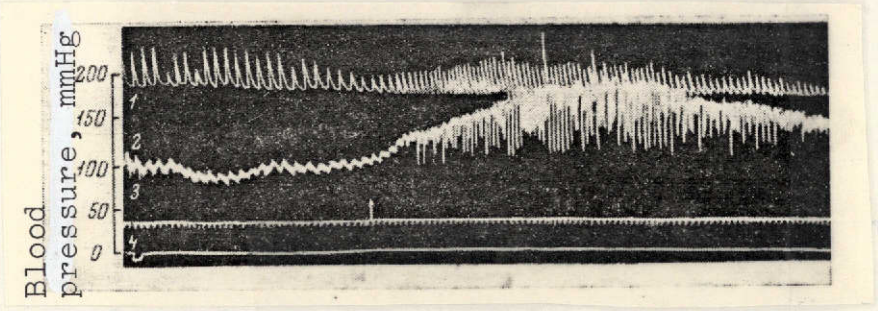

Fig. 48. Changes in respiration and blood pressure in dog incident to pronounced decompression aeroembolism. Start of recording, 12 min after decompression (pressure and exposure: 5 psig, 20 min; 4 psig, 2 h; 3 psig, 1 h 30 min; 4 psig, 11 h 30 min); 1 -- respiration; 2 -- blood pressure; 3 -- time indication, 3 sec; 4 -- indication of insertion of arterial gas trap. The arrow points to the moment of appearance of bubbles in the arterial blood.

These experiments are, as it were, a direct indication that incident to especially intense decompression effects,

aeroembolic phenomena develop not only in the venous, but also in the arterial, system.

Such a categorical conclusion, however, would be somewhat premature. In describing this research method, we pointed out that gas traps suffer from the shortcoming that the blood flow in them is appreciably slowed down. For the rapidly flowing arterial blood, this slowing down is particularly considerable. Inasmuch as the formation of a gas bubble that is visible to the naked eye takes a certain time, which can apparently be counted in at least tenths of a second, the thought arises that those gas bubbles which we see in the arterial gas traps may have managed to grow to this size in the process of the blood being slowed down through the instrument and that in undamaged vessels, the case may be somewhat different. Thus, the only completely categorical conclusion that can be drawn from experiments with arterial gas traps is that incident to pronounced caisson phenomena, the arterial blood presents all the conditions for the formation of many gas bubbles that are X172 visible to the naked eye, i.e., rather large ones. It is difficult to say at what stage this possibility arises in the intact organism. Indirect data obtained from the just-described experiments compel the assumption that intraarterial gas formation, though it does not decide the matter, does play a certain role in functional changes incident to decompression disorders. The experiments in which the appearance of numerous gas bubbles was detected in an arterial gas trap differed from the other experiments by certain distinctive features of the reaction of the cardiovascular system.

While bubbles are being formed in the arterial gas trap, the animals display a pronounced pressor reaction that considerably exceeds in intensity and duration the initial pressor effect detected in experiments where gas formation took place only in the venous blood. As can be seen from the typical kymogram of Fig. 48, though these blood pressure shifts coinciding with the formation of gas bubbles in the arterial gas trap are very considerable, they are neither catastrophic nor so sharp as to lead us to suppose that they are based on an aeroembolism of the cerebral arteries in the region of the cardiovascular center. Moreover, they are generally transient; in particular, in the experiment to which the kymogram belongs, decompression disorders did not lead to the dog's death at all; it was killed two hours 20 minutes after the end of decompression. This, too, argues against the centrogenic embolic nature of the pressor reaction we are talking about. It must be assumed that the rise in blood pressure, as in all the other cases, is connected with gas formation at the periphery of the vascular system and, above all, in the precapillaries, capillaries and venules of tissues particularly rich in nitrogen, especially adipose tissues. This gas formation in itself, causing local vascular spasms as it

does, leads to an increase in peripheral resistance, which is what causes the pressor effect. The particular degree of evidence and duration of this effect in those cases where gas formation is detected in the arterial gas trap is obviously explained by the fact that arriving at the periphery with the arterial blood are gas bubbles that have already been formed but have not, presumably, yet reached embolic size. There, in tissues rich with nitrogen, they encounter all the conditions for rapid growth, accelerate the process of gas formation and, consequently, intensify also all of its consequences involving the hemodynamics.

To study the possibility of postdecompression gas formation in the arterial blood, we undertook, in addition to the experiments on dogs that we have just described, experiments on 22 rabbits, in which we kept watch for the appearance of gas bubbles in different vessels. The animals were kept in the pressure chamber under an air pressure ranging from 2.25 to 5 psig for six hours. Decompression was effected either at the fastest possible rate (10-15 sec) or at a somewhat slower tempo (50-60 sec). In the first case, in order to avoid barotrauma of the lungs, a tracheotomy was performed on the rabbits 30 min-1 hour before the end of exposure. /173

In some experiments (nine rabbits), the animals were immediately killed with an electric current at the end of decompression. In others (13 rabbits), they were anesthetized with ether or an intravenous injection of hexanal, after which we subjected the killed animals to autopsy and the anesthetized ones to vivisection and inspected the vessels of the subcutaneous cellular tissue, the extremities, the organs of the abdominal cavity, the abdominal aorta and the rear vena cana and, in the corpses, the coronary vessels and the heart cavities, too. At the same time, we kept watch for the formation of gas bubbles in the adipose tissues that were accessible to observation.

In the rabbits killed and opened immediately after decompression (pressures of 2.25, 2.5, 2.75, 3.0, 3.25 and 5 psig, an exposure of 6 hours), in all cases without exception we noted the formation of gas bubbles in the veins as well as the arteries. The quantity of gas bubbles depended on the pressure employed: at 2.25 psig they could be easily counted in one or another of the vessels; for every fourth of an atmosphere of increasing pressure their number increased; and at 3.25 psig there were a great many of them; finally, at 5 psig the vessels proved to be completely filled with gas in a short while.

At pressures of 3.25-2.25 psig the first gas bubbles appeared in the vessels in 3-5-10 min after decompression, while at a pressure of 5 psig they were detected immediately after autopsy. The increase in the number and volume of the gas bubbles and gas accumulations being formed took place very

intensively in the first 20-30 min after decompression but also continued subsequently, sometimes in the course of the entire period of observation, which took 1.5-2 hours. It was obvious, especially in experiments with pressures of 3, 3.25 and 5 psig that with the passage of time, the arterial as well as venous vessels were filling with gas from the periphery. Thus, it sufficed to make a massaging motion along the course of the femoral vessels from the periphery of the extremity toward the body for the prepared proximal sections of the femoral artery and vein to become immediately overfilled with gas bubbles and gas accumulations. In these, as well as in the mesenteral vessels, it could be seen that even without any mechanical effects, the gas bubbles and accumulations that had been formed in the arteries and veins were gradually moving away in a central direction, forced back by new batches of gas being formed at the periphery. Evidently, even when the blood flow is arrested, nitrogen diffusion takes place from the tissues into the gas bubbles being formed in the capillaries.

As to the intensity of gas formation in the experiments under discussion, it was difficult to ascribe preference to the vessels of one or another region; it is important to stress that nowhere was there any great difference between arteries and veins. In individual experiments, gas formation in the arteries began even earlier and proceeded more intensively than in the corresponding veins. To exclude the possibility of gas bubbles passing through the arteriovenous anastomoses, in a number of experiments we isolated individual sections of the arteries and veins with ligatures. In all cases where the isolated sections of vessels were extremely small, gas formation was observed both in the arterial and the venous blood. All these facts are quite understandable. For the short time of decompression and the 15-30 seconds that elapsed at the surface until the animals' death, there could be no significant desaturation, and all the tissues of the organism, as well as the arterial blood, proved to be supersaturated with nitrogen to an equal extent. Also, if we proceed from the fact that the arterial blood gets into diffusion equilibrium with the alveolar air through nitrogen as rapidly as it does through oxygen and carbon dioxide, i.e., practically instantaneously, then under these conditions one might have expected somewhat less gas formation in the arterial blood as compared with the venous blood, but the experiments did not show this. /174

In the rabbits that were anesthetized and subjected to vivisection immediately after decompression, gas formation in the venous blood was also observed in all cases without exception. These experiments differed from the preceding ones in the fact that the gas bubbles appeared somewhat later, in a smaller amount and preferentially in veins bearing blood from tissues rich in fat.

When the blood is moving, it is not possible to observe gas bubbles in the arteries. In this connection, in the anesthetized animals, we undertook ligation of the artery at different times after decompression. In some cases, we ligated the abdominal aorta under the diaphragm, simultaneously ligating the vena cava at the same level and thus arresting the blood flow throughout the rear part of the body. In other cases, we ligated the femoral and renal arteries and veins in different places, as is shown in Fig. 49, and ligated the ramifications of the mesenteral arteries and veins.

Regardless of the type of ligature of the different vessels, in almost all the experiments we isolated between two ligatures small branchless sections of the mesenteral arteries (Fig. 49, 3) and, in a number of experiments, small branchless sections of the abdominal aorta.

The experiments showed that when we ligated in the first 10-15 min after decompression, gas bubbles always appeared in the ligated arteries, including their completely isolated sections. Being small at first, these bubbles gradually became larger, growing in a number of cases into voluminous gas accumulations. The first point bubbles that were scarcely perceptible to the naked eye appeared in the arteries not at once, but on expiration of a few dozen seconds or even 1-1.5-2 min. The further growth /175 of the bubbles at first proceeds more rapidly, then more slowly, but generally extends over a very long time; for the first 10-20 min, the point gas bubbles grow into large bubbles or still more massive gas accumulations; but in either case, their volume generally continues to increase for several dozens of minutes. Consequently, it is not that the ligature of arteries and the cessation of the blood flow in them permit gas bubbles to appear that were formed earlier in the circulating arterial blood; the matter undoubtedly reduces to gas formation in the blood of the isolated arterial sections. True, the experiments with the gas traps compelled the hypothesis that in the circulating arterial blood incident to pronounced caisson phenomena, there may always be a rather great number of microscopic "gas seeds." But even under this condition, the fact that new large quantities of gas are formed in the blood of the ligated arteries cannot be doubted.

Intraarterial gas formation turns out *ceteris paribus* to be the more intensive, the earlier the blood flow is arrested, while if the ligatures are effected in the first minutes after decompression, the intensity of gas formation in the arteries is generally even somewhat greater than in the corresponding veins ligated in a similar manner. At a pressure of 3.25 psig, a 15-minute period after decompression turned out to be the limit for intraarterial gas formation: when arteries are ligated after this period, gas bubbles never arise in them. But /176 bubbles can still be observed to form in unligated as well as ligated veins for dozens of minutes after this.

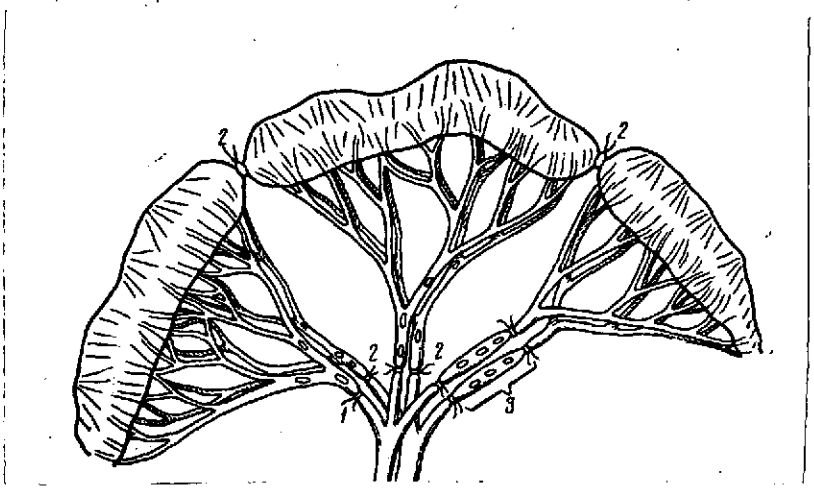

Fig. 49. Different types of ligations of mesenteral vessels. 1 -- ligation of regional artery and vein; 2 -- same, with ligation of the intestine by two ligatures circumscribing the region that supplies blood through the vessels in question; 3 -- ligation by dual ligatures of sections of mesenteral arteries and veins in places having no branches.

Unexpected was the fact that gas bubbles and gas accumulations appear in the arrested arterial blood when ligatures are applied for a comparatively long time (at a pressure of 3.25 psig, up to 15 min) after decompression. It is customarily assumed that diffusion equilibrium between the blood flowing through the pulmonary capillaries and the alveolar air is established for inert gases almost as rapidly and completely as for oxygen and carbon dioxide. Even if we take into account the relatively lesser diffusibility of nitrogen as compared with oxygen and carbon dioxide, a period of 10-15 minutes after the termination of decompression should nevertheless be quite enough for the blood flowing through the pulmonary vessels to rid itself of excess nitrogen, if not completely, then at least to such a degree of supersaturation at which gas bubbles are not formed. This might have been expected, the more so, as the pressures employed in our experiments and, consequently, also the degree of supersaturation with nitrogen of the organism as a whole and so of the blood, too, were relatively low. In spite of this, experiments showed that even 15 minutes after decompression, distinct, quite often very intensive, gas formation could be observed in the arterial blood, if its movement was stopped.

So a new question arises: are not prevailing ideas about the rapid diffusion of nitrogen through the alveolar membrane,

as well as through the walls of tissue capillaries, erroneous? Is it not rather that, after decompression, the venous blood supersaturated with nitrogen reaching it from the tissues is able, while flowing through the pulmonary capillaries, to give off to the alveoli only a small part of this gas and passes into the arterial system still supersaturated with it to a considerable extent?

In such a case it would be understandable that the supersaturation of the arterial blood could decrease to safe levels only when the excessive supersaturation of the venous blood decreases correspondingly and that this, in turn, could occur only when the degree of supersaturation of the tissues decreases, i.e., when the organism as a whole is desaturated to a certain extent from the nitrogen dissolved in it in excess. Could it be that the 15-minute period in our experiments reflected such a degree of desaturation of the organism? In connection with this line of reasoning, the thought arises whether it is not the retarded diffusion of nitrogen that explains the well-known circumstance that for a man's stay at every depth there exists a safe time, i.e., a period of action of increased pressure after which a rapid ascent to the surface can still be effected with no fear of decompression disturbances. /177

With regard to the reason for gas formation in the arrested arterial blood for considerable periods of time after decompression the following two assumptions can also be made.

In the first place, it can be assumed that the long-enduring supersaturation of the arterial blood after decompression is determined by the fact that what is slowly desaturated is not the plasma, but formed elements of the blood. That is to say, we can imagine that as the blood flows through the pulmonary capillaries, the plasma succeeds in becoming desaturated to levels that are safe from gas formation, but inasmuch as nitrogen contained in formed elements keeps on passing into it, its supersaturation again becomes excessive.

In the second place, it can be assumed that in the formation of intraarterial gas bubbles incident to arrested blood flow a role is played by the diffusion of nitrogen into the blood from artery walls. Being rich in lipoids, artery walls must dissolve a considerable amount of nitrogen, and in those cases where, owing to the long action of increased pressure, they are completely saturated with nitrogen, their desaturation after decompression must take no little time.

Thus, the reasons for gas formation, incident to standstill of the blood flow, in the arteries for a considerable time after decompression can be reduced only to three circumstances: owing to the slow diffusion of nitrogen in the lungs, the arterial blood as a whole remains supersaturated with it for the indicated

periods after decompression, or the providers of the nitrogen are formed elements that are desaturated more slowly than the plasma, or the nitrogen diffuses into the arrested blood from artery walls.

It goes without saying that all three of these possible reasons do not exclude one another and can act simultaneously.

Be that as it may, the results of experiments leave no doubt that in the indicated cases (rapid decompression after a 6-hour exposure to a pressure of 2.5-3.25 psig), all the conditions for gas formation exist in the arterial blood (in the arterial vessels) for 10-15 min after decompression.

Obviously, the only reason that these conditions are not realized in the intact organism is the rapidity of flow of the arterial blood. As has already been stressed, for bubbles that are visible to the naked eye to be formed in a supersaturated solution takes several minutes, while a certain amount of the arterial blood makes it entire way from the pulmonary capillaries to even the furthest regions of the body in a matter of seconds. This time is clearly insufficient for the microscopic gas bubbles passing through the pulmonary capillaries, or the gas "seeds" arising in the arterial blood itself, to grow to large sizes. These facts make it necessary to approach the question of decompression aeroembolic phenomena in the arterial system /178 in a somewhat novel manner.

First of all, that alternative formulation of the question of the permeability of the pulmonary capillaries to gas which is adhered to by authors who have examined this question as applied to venous aeroembolisms of nondecompression origin, cannot be correct for cases of decompression aeroembolism. Familiarity with the sources in the literature shows that this question has generally been raised as follows. If the pulmonary capillaries are permeable to gas bubbles, as is believed by Sotakis, Lermitt and Gassen', Pines (quoted by M. I. Yakobson, [1950]), then the appearance of gas bubbles in the arterial blood is quite possible when air gets into the veins; if, on the other hand, the pulmonary capillaries are impermeable, as most researchers assert (Shestopal, 1898; Miram, 1909; Il'in, 1914; Curtiliet, 1939; etc), then there ought to be no gas bubbles in the arteries. This alternative, as it seems to us, misses the point. The experiments that we carried out have convinced us that the pulmonary capillaries, in contrast to the vascular network of the greater circulatory system, do not let pass air bubbles that are visible to the naked eye; but this does not at all mean that they are absolutely impermeable to microscopic air emboli. The latter, after plugging up the capillaries, decrease in volume owing to the diffusion of nitrogen into the alveolar space and become smaller than the lumen of the capillary.

At the same time, they must inevitably be carried into the pulmonary veins by the force of the blood's pressure head.

If the arterial blood is not supersaturated, the microscopic gas bubbles, as A. P. Brestkin [1952] has pointed out, may become dissolved in the plasma owing to the overpressure in them, which increases as they decrease in volume. Obviously, owing to this self-destruction of the subcapillary gas bubbles passing through the pulmonary vascular network, we could find no air in the arteries after injecting it into the veins.

The case of decompression aeroembolism is different. Here, as experiments have shown, in the course of several minutes after decompression the blood remains supersaturated with nitrogen, and all the conditions exist for the growth of the gas "seeds" passing through the pulmonary capillaries. However, owing to the high flow rate of the arterial blood, the gas bubbles in it do not succeed in growing to large sizes and, as a rule, no aeroembolism of the peripheral arteries of the greater circulatory system takes place; the more so, as the peripheral vascular network, in contrast to the pulmonary one, is readily permeable to gas bubbles.

It by no means follows from all of the foregoing that we can disregard the formation of gas bubbles in the arterial blood, even those of subembolic size. Such bubbles, as will be shown below, can play a very important role in the development of decompression disorders.

The results of the experiments described in the present chapter permit us to draw the definite conclusion that the main link in the development of decompression disturbances is aeroembolism of the venous system and pulmonary capillaries. This is directly borne out by the following: the character of the reactions of the respiration and blood circulation incident to the development of caisson phenomena; the direct dependence between the dynamics of these functions (as well as the degree of change in them) and the intensity of gas formation in the venous blood; finally, the possibility of reproducing the picture of decompression disturbances in all of its essential details by intravenous injection of air. On the basis of all of these facts, the development of decompression aeroembolisms can be imagined in the following manner. 179

Immediately after decompression (and, if it is dragged out in time, in the very process of decompression) the excess nitrogen dissolved in the tissues begins to diffuse into the blood reaching them. As soon as the coefficient of maximum permissible supersaturation of the blood is exceeded (and, as A. P. Brestkin has shown, it is considerably lower than the corresponding coefficient for the tissues), gas bubbles

begin to form in it. The nitrogen pressure in the blood reaching the tissues increases as the blood flows through the capillary and attains a maximum in its venous end. Therefore, the venous parts of the capillaries and the venules are the place where gas bubbles are most readily formed. The nascent gas bubbles in the capillaries and venules, moving along together with the blood, flow into the fine, and then the larger, veins, increase in size and merge with one another, forming still larger bubbles or gas accumulations, which get into the main venous vessels and head for the heart together with the blood.

The fact that the gas bubbles and gas accumulations that we see in the venous blood are formed mainly on account of the merging of a great number of microscopic gas bubbles that cannot be seen by the naked eye on their way from the capillaries to the large veins, is beyond any doubt. In the first place, as has already been repeatedly stressed, it takes a certain amount of time for a gas bubble to grow from an infinitesimal size to a size that can be seen by the naked eye, a time that undoubtedly exceeds the time it takes the blood to flow from the capillaries to the large veins. In the second place, if we assumed that each capillary bubble grows to a visible size, then, taking into account the huge number of capillaries, we would have to acknowledge thattin this case, the veins could not accomodate the entire amount of gas being liberated. The merging of gas bubbles with one another continues, as can already be seen by the naked eye, in the small, as well as larger, veins; here it is clearly noticeable how gas bubbles simultaneously arriving at the place where two venous trunks merge, coalesce, forming larger gas accumulations. Finally, the recombination of individual gas bubbles with one another can be observed in the main venous /180
vessels, too; for example, in the femoral, jugular and other large veins. Thus, incident to pronounced aeroembolic phenomena, the gas set free from solution gets into the heart to a considerable extent in the form of more or less large gas bubbles and whole gas accumulations, though apart from them the blood contains also a great amount of finer gas bubbles that did not manage to grow, including, one must suppose, such as are not visible to the naked eye. In the heart, owing to its contractions, the reverse process begins -- the breaking down of gas accumulations and large gas bubbles intoofiner ones. Observation is convincing proof of this. Opening the thorax of animals during life (in the case of artificial respiration) or after death and examining the vessels, in the venae cavae we always find, incident to intensive caisson phenomena, large gas bubbles and gas accumulations, while in the pulmonary arteries we see only numerous fine bubbles, or, incident to a particularly intensive aeroembolic process, finely frothing blood. In cats and, especially, rabbits, through the fine wall of the right auricle, one can see that here, in the atrium, gas accumulations are already beginning to break up. This can be seen even more clearly upon opening the cavities of the right side of the

heart and the pulmonary arteries: the outflowing blood contains only fine bubbles or, in serious cases, the gas gets mixed with the blood and forms a froth; right after the animal's death, large gas accumulations are never present here; they can be formed only on expiration of a more or less long time after cardiac arrest.

The breaking down of gas bubbles that begins in the cavities of the right side of the heart undoubtedly continues in the ramifications of the pulmonary arterial vessels, too, right up to the pulmonary capillaries. One must suppose that, contrary to what happens in the venules and veins of the periphery of the body incident to the merging of gas bubbles, in the lungs these bubbles, on approaching the next arterial ramifications, split up and disintegrate into ever finer ones.

The breaking up and fragmentation of gas accumulations and gas bubbles that takes place in the heart and pulmonary vessels is a special process, of extreme importance for the entire course of aeroembolic phenomena. If this did not happen and if this process did not begin as early as the heart, the gas accumulations and large gas bubbles leaving it for the pulmonary arterial vessels, on flowing through the arterial ramifications, would sooner or later create in them such long gas plugs that the blood circulation in the corresponding vessels would stop sooner than air could reach the pulmonary capillaries -- the place where air emboli are resorbed and liquidated. The matter would therefore reduce to total aeroembolism not of the end capillaries, but of the larger ramifications of /181
the arterial vascular network and to a block of blood circulation with no possibility of liquidating the gas bubbles.

The breaking up and fragmentation of gas bubbles is extremely important for still another reason. We have already referred to A. P. Brestkin's hint that with decreasing volume of the gas bubble, the pressure within it increases in conformity with Laplace's equation, this increase in internal pressure taking place particularly abruptly when the bubbles decrease to microscopic size. If the overpressure in large gas bubbles, which counteracts the forces of surface tension and ensures that the bubbles will pass into solution, can be counted in mmHg, then in the point bubbles that are scarcely visible to the naked eye, it will already amount to tens of millimeters; in finer bubbles that are inaccessible to observation, to fractions of an atmosphere; and in microscopic bubbles to whole atmospheres.

The internal pressure in the bubble is a factor that causes the gas to diffuse from the bubble into the surrounding fluid medium, i.e., a factor that is conducive to a reduction in size and liquidation of the bubble.

It can be seen from the adduced figures that in small bubbles, this factor acquires considerable importance and the more so, as the bubble's volume is less. Microscopic bubbles, as A. P. Brestkin has pointed out, are prone to self-liquidation: under the influence of the internal overpressure, the gas molecules from the bubble diffuse into the surrounding fluid, the bubble decreases in volume, with decreasing volume the internal pressure increases and, consequently, diffusion is intensified, and so forth until liquidation of the bubble. Figuratively speaking, gas bubbles that have decreased to microscopic size are, as it were, more and more squeezed by the surrounding fluid and, in the end, dissolve completely. Of course, none of this can take place unless there is a certain pressure in the fluid medium of that gas out of which the bubble is formed.

When the fluid is supersaturated with this gas in excess of the coefficient of maximum permissible supersaturation, the reverse takes place: the forces of diffusion, under whose influence the gas bubbles go over from solution into nascent gas "seeds," overcome the force of surface tension acting at the periphery of the bubble, and lead to its growth, just as happens incident to the formation of bubbles in the peripheral venous blood. In the case of decompression effects that do not lead to the animal's rapid death, at a certain stage in the development of caisson phenomena, conditions must inevitably be created under which the pressure of the nitrogen in the total venous blood proves to be less than in the peripheral venous blood of those regions where gas bubbles are still being formed. This is how matters will always stand in those cases where a number of organs and tissues that possess less stores of nitrogen or are distinguished by energetic blood circulation, are /182 already to a considerable extent desaturated, while others (for example, adipose tissues) are still rich in nitrogen and the blood in their capillaries is still being supersaturated in excess of the coefficient of maximum permissible saturation and gas bubbles are being formed.

The nitrogen pressure in the total venous blood will in this case always be less than in the venous blood flowing out from the adipose tissues, and its supersaturation may be below the maximum permissible one.

Under such conditions, the fragmentation of gas bubbles taking place in the heart and pulmonary arterial vessels can lead to liquidation of some of them before they reach the pulmonary capillaries. But there can be no doubt that this liquidation of air emboli, if it does indeed take place under certain conditions, never acquires decisive significance. The great mass of air emboli are retained by the pulmonary capillaries. Here it is that the organism is freed from the gas that formed in the blood: it diffuses into the alveolar space from the

gas bubbles that had been plugging the capillaries. As soon as a gas bubble in one or another capillary decreases, owing to this diffusion, to a size less than the lumen of the capillary or vessels close to it, under the blood's pressure head it must pass into the pulmonary veins, i.e., into the arterial blood. The subsequent fate of these microscopic gas bubbles passing through the pulmonary filter will be entirely determined by the extent to which the arterial blood remains supersaturated with nitrogen and whether conditions in the arterial vessels are favorable to gas formation. If supersaturation is low and no such conditions exist, the microscopic bubbles are foredoomed to self-dissolution and liquidation. If supersaturation is considerable and such conditions do exist, which, as experiments have shown, may be so for many minutes after decompression, the microscopic bubbles in the arterial blood not only will not be liquidated, but will begin to grow. But, owing to the high flow rate of the arterial blood, they do not succeed in growing to a considerable size and there will generally be no aeroembolism of the peripheral arteries.

Thus, the development of aeroembolism incident to considerable supersaturation of the organism in the early period after decompression can be pictured in the following manner. The great masses of gas that are constantly being formed in the venous blood are retained in the pulmonary capillaries, where nitrogen diffusion takes place from the air emboli into the alveolar space. At the same time, microscopic gas bubbles penetrate into the arterial blood through the pulmonary vascular network; owing to the supersaturation that exists here, they begin to grow; but, failing to attain a large size because of the rapidity of the blood flow, they get into the capillaries of the tissues, where it is that they encounter all the conditions for a subsequent increase in volume. Thus, ever newer batches of gas are /183 brought to the lungs, where they are retained and to a considerable extent liquidated, while only the finest gas bubbles constantly pass through the arterial system. Consequently, what emerges as the process determining the physiological reactions of the organism and its condition in these cases is aeroembolism of the venous system and pulmonary capillaries.

But it would be rash to conclude that the possibility of gas formation in the arterial blood, which arises under certain conditions and which persists, as experiments have shown, for a considerable time after decompression, does not threaten the organism at all, or that the presence of microscopic gas bubbles in the arterial vessels in no way influences the development of caisson phenomena.

Intraarterial gas bubbles, even if they do not attain embolic size, must change in a substantial manner the process of gas formation in the peripheral capillary network. In order to get a clearer idea of this, let us turn to the simplified diagrams

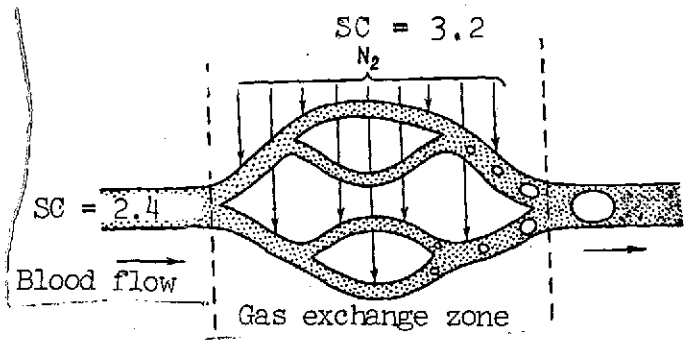

Fig. 50. Diagram showing gas formation in capillaries of the greater circulatory system that supply adipose tissues, in the absence of gas bubbles in the arterial blood. The arrows indicate nitrogen diffusion from the tissues into the blood; SC is the coefficient of maximum permissible supersaturation (for blood, 2.4; for adipose tissues, 3.2)

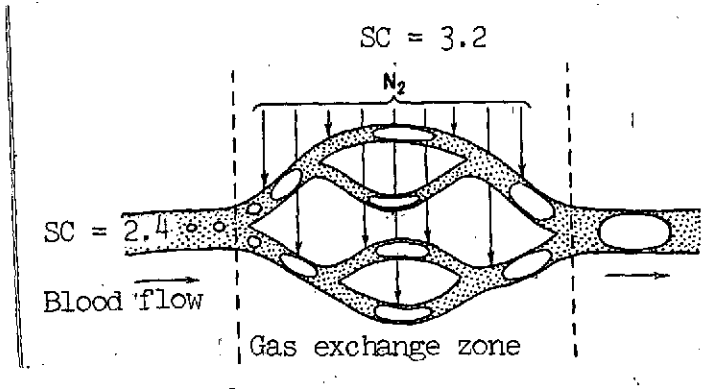

Fig. 51. Diagram showing gas formation in capillaries of the greater circulatory system that supply adipose tissues, in the event of supersaturation of the arterial blood with nitrogen and the presence of gas bubbles in it. Same notation as in Fig. 50.

of Figs. 50 and 51. Fig. 50 represents the case where the arterial blood getting into the tissue capillaries is not supersaturated with nitrogen and, consequently, contains no gas bubbles or gas "seeds." In this case, as has already been pointed out, the pressure of the nitrogen diffusing into the blood that is slowly flowing through the capillary will gradually grow in it and will attain a maximum when the blood passes into the venules. It is precisely here, consequently, and by no means in the beginning part of the capillary, that gas bubbles will be formed in the given case. Their retention in the place where they are formed is not very likely inasmuch as the blood from the capillary passes into wider venules and then into the veins, whereupon its flow rate increases sharply.

But the case is different /184 (Fig. 51) when arterial blood supersaturated with nitrogen and containing gas bubbles, even of subcapillary size, arrives at the tissues. Inasmuch as the arterial blood is supersaturated, these bubbles, regardless of the blood's motion, grow with a certain speed and their increase in volume is a direct function of time. When the blood passes from the fine arteries into the arterioles and capillaries, an extremely abrupt slowing down of the blood flow takes place. The flow rate of the blood in the capillaries is 400 times less than in the aorta [Ginetsinskiy and Lebedinskiy, 1947]; but the arterioles and precapillaries are those sections along which, on the whole, a decrease in speed takes place. Consequently, the finest gas bubbles contained in

the arterial blood will grow and manage to increase in volume before the blood gets into the capillary -- into the zone of gas exchange with the tissues. But here they will at once meet still more favorable conditions for their growth on account of the diffusion of nitrogen from the tissues and the maximally slowed down blood flow. In the present case, in contrast to the preceding case, the gas will be liberated not only in the venous part of the capillary or in the venules, but as early as the very beginning of the capillary and even before it. A temporary and even persistent retention of gas bubbles in the capillary network under such conditions is quite possible and probable, especially if we take into account the possibility that, as was pointed out by V. V. Pashutin [188], the terminal ramifications of the arterial vessels may undergo reflex spasms in response to the bubbles existing in them. An indirect argument for the actual occurrence of such a retention is the pronounced and prolonged rise in the arterial pressure in those cases where formation of gas bubbles is noted in the arterial blood (in the gas trap). This pressor effect, which is monotypic in character for all such cases, is apparently connected, as was already mentioned above, with an increase in resistance of the blood flow as a result of intensified gas formation in the peripheral vascular network.

Significantly, the intensified gas formation in the capillaries and the retention of gas bubbles there can cause a slowing down of the blood flow in the corresponding ramifications of the arterial network, and this, in the event of supersaturation of the arterial blood with nitrogen, will lead to a growth of the gas bubbles in the arterial blood to a greater size even before the blood gets into the capillary network.

So the matter may resemble a vicious circle and terminate in aeroembolism of the corresponding arteries with complete standstill of the blood flow in the region in question.

Thus, depending on the degree of supersaturation of the arterial blood with nitrogen and whether it contains gas bubbles, if only microscopic ones, the process of gas formation in the peripheral vascular network will shift either toward the veins or toward the arteries. In the first case, local disorders of the blood circulation are less likely, and in the second case, more likely. But in all cases the initial cause of these local hemodynamic disorders is gas formation in the capillaries, the intensity of which is determined by the degree of supersaturation of the corresponding tissues with nitrogen (or some other inert gas).

The idea that the initial cause of local disorders of the blood circulation is obstruction of the ramifications of the arterial vessels with air emboli does not correspond to the

facts. In this respect, decompression aeroembolism differs essentially from the other embolisms encountered in clinical practice.

There, the danger of local disturbances of the blood circulation in any organ is determined by the probability that embolizing particles will accumulate in its arterial vessels. Incident to decompression aeroembolism, this danger is determined by the degree of supersaturation of the given organ's tissues with an inert gas and the intensity of formation and possibility of retention of gas bubbles in its capillary network. That is precisely why the symptoms connected with local disturbances of the blood circulation in the brain are encountered incomparably more rarely in decompression aeroembolism than in arterial embolisms of any other etiology. The extremely intensive blood supply of the brain ensures its rapid desaturation from an inert gas after decompression, which decreases the probability of formation and retention of gas bubbles in the cerebral capillaries.

It is evidently for the same reason that embolism of the coronary arteries, which is very frequent in clinical pathology, is generally not encountered in decompression disturbances as an independent syndrome and can only accompany total aeroembolism of the entire vascular system incident to excessively severe /186 decompression effects. It follows from the foregoing that those cases of caisson phenomena in which the arterial blood proves to be supersaturated with nitrogen and contains microscopic gas bubbles present a particular danger for the well-being of individual organs, as well as for the well-being of the organism as a whole. In such cases, the organism stands every minute on the threshold of serious and even fatal disturbances.

A short-lived slowing down and, even more so, an arrest of the blood flow under such conditions can have fatal and irremediable consequences. This is illustrated with the greatest clarity by special experiments that we conducted on rabbits. The animals were kept under a pressure of 10 psig (a depth of 100 m) for that maximum permissible length of time after which rapid (for 100 sec) decompression to normal pressure threatens them with serious decompression disturbances. This length of time for fresh rabbits amounted to 5-6 min and, on repeating the experiments, in view of the animal's conditioning, increased to 8-9 min.

The experimental conditions, as is borne out by experiments with gas traps, were undoubtedly such that in the early period after decompression, the venous, as well as the arterial, blood of the animals proved to be supersaturated with nitrogen. In the experiments, some animals served as controls and were not subjected to any additional effects; in others, during the first 15 min after decompression, reflexes were evoked from the upper respiratory tract for different lengths of time by means of

forced inspiration of vapors of an aqueous solution of ammonia. A piece of cotton was soaked in the solution and held against the rabbit's nose for no more than 8-10 sec. This, as is well-known, results in apnea and inhibition, and sometimes also in short-lived cardiac arrest. Both the apnea and the inhibition of heart action lasted after the 8-10 seconds of stimulation for no longer than the same length of time, i.e., their total time did not exceed 15-20 sec. These reflexes, which are defensive in character and quite harmless for normal rabbits, proved to be fatal for animals emerging from under increased pressure. Usually 15-20 sec after respiration and normal heart contractions were restored, now irreversible disorders of them redeveloped, the rabbit fell on its side and began to thrash about in spasms; the latter rapidly changed into a general tonic extensor spasm -- acute opisthotonos accompanied by the animal's squeal -- and death set in. With the spasms, there developed exophthalmos, pupil dilation, nystagmus, salivation, often defecation and urination. The entire picture of functional disorders and death of the rabbits bore witness to the rapid and irreversible disturbances of the cerebral and, probably, total blood circulation. In these experiments, 13 out of 15 /187 rabbits died, whereas only three out of 15 controls died.

The results of these experiments can only be interpreted to mean that even short-lived slowing down or cessation of blood flow and stoppage of respiration caused by a reflex from the upper respiratory tract led to sharp intensification of aero-embolic phenomena as a whole and, in particular, to gas formation in the arterial blood, i.e., to rapid growth of the microscopic gas bubbles existing in there to a large embolic size.

Hence, it follows that in all cases where not only the venous, but also the arterial, blood is supersaturated with nitrogen, the organism's struggle with aeroembolic phenomena goes beyond its compensatory resources. From this and all of the foregoing it also follows that these compensatory resources for all cases of decompression disturbances are determined by the state of the respiration and of the blood flow.

Thus, there is every reason to suppose that the increase in the organism's resistance to decompression disturbances, as decompression effects are repeated and gradually intensified, is connected with compensatory reactions of the respiration and of the cardiovascular system in response to aeroembolic phenomena. Everything that has been said leads to the conclusion that the changes in respiration and cardiovascular activity incident to the development of caisson phenomena in anesthetized dogs reduces to panting similar to that of hypoxemia, to initial tachycardia accompanied by a short-lived pressor effect and to subsequent bradycardia and a drop in blood pressure. Fatal cases of decompression disturbances are characterized by a steady

drop in blood pressure, right down to critically low levels, which take place against a background of progressive increase in pulse rate and panting; incident to the animal's death, respiration, as in the case of fatal hypoxemia, ceases earlier than heart action.

The beginning of the changes in respiration and blood circulation in animals after decompression coincides with the appearance of gas bubbles in the veins; the first single gas bubbles in the venous blood appear after the animals have stayed at a depth of 12 m. The character and extent of the changes in respiration and blood circulation incident to the development of decompression disturbances after emerging from the depths are directly connected with the intensity of gas formation in the venous blood. This testifies to the leading role of aeroembolism of the the venous system in the development of decompression disturbances.

The changes in respiration and blood circulation arising in animals incident to intravenous injection of air and helium are very close to the changes in the functions incident to pronounced decompression disturbances. This again confirms the leading role of aeroembolism in the development of decompression disorders.

The organism displays exceptional resistance to artificial /188
and to postdecompression aeroembolism; incident to the development of the latter, it can keep alive when the peripheral veins are overfilled with gas bubbles and there is a huge number of them in the total venous blood.

Upon being injected intravenously, different gases differ sharply in the intensity of their physiological effect. The organism endures the intravenous injection of nitrogen least easily of all, helium considerably more easily, oxygen still more easily and carbon dioxide especially easily. This is explained by the different diffusibilities of the enumerated gases, and for O_2 and CO_2, also by the possibility of their chemical bonding in the organism.

The consequences of artificial aeroembolism depend to a great extent on diffusion relationships accumulating in the lungs between air emboli and the gaseous medium filling the alveolar space. Hence it follows that in the liquidation of air emboli, a leading role is played by the diffusion of the gas composing them from the pulmonary capillaries into the alveoli.

The pulmonary capillaries do not let pass gas bubbles that are visible to the naked eye, whereas the peripheral ramifications of the vessels of the greater circulatory system prove to be readily permeable to gas.

When the arteries of rabbits are ligated after their prolonged stay under a pressure of 2.25 psig and higher, for the 10-15 min after decompression gas bubbles and large gas accumulations are formed in the completely isolated sections of the arterial vessels; incident to intensive postdecompression gas formation in the venous blood of dogs, gas bubbles can be seen to appear in gas traps inserted in their arteries, which is accompanied by a pronounced pressor reaction and bradycardia. The growth of the gas bubbles in the arterial gas trap to a size that is visible to the naked eye is apparently connected with the slowed down flow of blood through the instrument.

It follows from the above-mentioned fact that when the organism is considerably supersaturated with nitrogen, all the conditions for gas formation exist in the arterial vessels for a comparatively long time after decompression (at 3.25 psig, up to 15 min, and at higher pressures, up to 30 min); but, owing to the high flow rate of the arterial blood, the gas bubbles in it do not, as a rule, succeed in growing to a size that can be seen by the naked eye. Under such conditions, the most short-lived slowing down of blood circulation and inhibition of respiration cause the rapid death of the organism, obviously because of gas formation in the arterial blood.

The obtained data suggest the idea of comparatively slow nitrogen diffusion in the lungs and, apparently, in the tissues, too.

CHAPTER 10

PHYSIOLOGICAL MECHANISMS OF THE INCREASE IN THE ORGANISM'S RESISTANCE TO DECOMPRESSION DISTURBANCES /189

At the end of the last chapter, the hypothesis was advanced that the increase in the organism's resistance to decompression disturbances incident to repeated decompression effects is connected with compensatory reactions of respiration and blood circulation that are evoked by the aeroembolic process. Although all of the material set forth above gives grounds for regarding this as being more than probable, it still does not allow this hypothesis to be accepted as an incontestible and obvious fact.

First of all, on the assumption that the increase in resistance to caisson phenomena is based on reactions to decompression aeroembolism, it was natural to check whether the organism is able to increase its resistance to artificial aeroembolism incident to its frequent repetition, i.e., whether a conditioning effect is possible here. The experimentally established similarity between changes in respiration and blood

circulation incident to artificial and decompression aeroembolism suggested that such an effect was very probable. In that event, it was logical to raise the following question: when the organism's resistance to artificial aeroembolism is increased, is its resistance to decompression disturbances increased at the same time, and, conversely, is the increase in resistance to decompression disorders that is caused by repeated decompression effects accompanied by an increase in resistance to intravenous injection of gases; the existence of such a conditioning effect would no longer leave it open to doubt that the increase in resistance to decompression disturbances incident to repeated decompression effects is connected with reactions to aeroembolism. It then became necessary to study these reactions of respiration and blood circulation in intact animals and trace how they change in the process of conditioning to artificial and decompression aeroembolism. After all of this, it was not possible to sidestep the question of the mechanism of these reactions. In this connection, the data obtained earlier also permitted certain assumptions to be made, namely, the similarity between the changes in respiration and blood circulation incident to decompression disturbances and the changes in these functions incident to hypoxemia, and the very fact of mass embolization of the pulmonary capillaries suggested the thought that the reactions to artificial and decompression aeroembolism are evoked by a disturbance of the gaseous composition of the arterial blood and, in particular, a drop in the oxygen pressure in it.

In the present chapter, an account is given of experimental /190 data on the above questions, none of which, except perhaps the last, has even been studied or even raised by investigators.

Conditioning of the organism's resistance to artificial aeroembolism

Experiments devoted to the present question were conducted jointly with A. S. Yurova on 32 rabbits. Of these animals, 16 were used in control experiments. In all cases, the air was injected into the rabbits' auricular veins from a well-ground syringe through a fine needle at a rate of 1 ml every 15 sec.

At the end of the injection, the animals without fixation were placed on a table and uninterruptedly observed for two hours. First we determined the air doses causing in the rabbits incipient, very transient functional shifts, the doses giving rise to pronounced disturbances and, finally, the doses leading to irreversible disorders and the animals' death. The first reactions in the form of slight panting and some general inhibition of the animal are detected after injection of 0.5-0.6 ml air. Doses of 1.0 ml cause pronounced panting, a general

depression of the animal and, sometimes, slight, very transient pareses of the rear extremities. Incident to injection of 1.3 mℓ air, the panting and general depression become even more pronounced, and opisthotonos and pareses of both rear extremities set in (Fig. 52).

As a rule, in the course of time all of these manifestations pass without leaving a trace; only in individual cases do the rabbits die. Doses of 1.5 mℓ prove to be fatal for the majority of the animals; in the agonal period, they develop general spasms. Finally, doses of 2.0 mℓ and more air prove to be absolutely fatal for the rabbits (Fig. 53). It should be stressed that even incident to injection of doses of air that are known to be fatal, at first almost all of the animals, in addition to general depression and sharp panting, develop paralyses of the rear extremities and the entire rear part of the body; the front extremities, if they become paralyzed at all, always do so later (Fig. 54).

Table 8 shows the results of the control experiments.

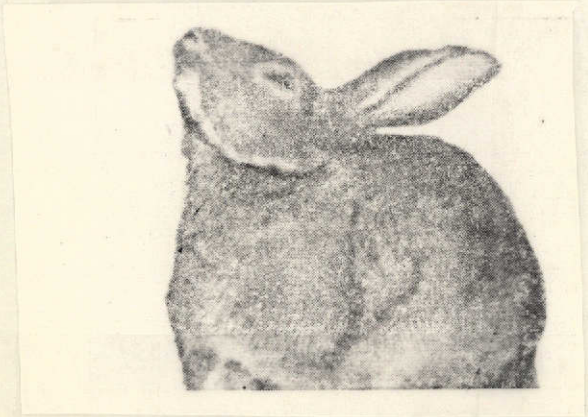

Fig. 52. Rabbit 3 min after intravenous injection of 1.3 mℓ air. Incipient opisthotonos.

Having determined fatal doses of air in the control experiments, we began our main experiments. The order of carrying them out was as follows. To evaluate initial reactions, each rabbit at first received 1.2-1.3 mℓ air. Then, for a number of days, the rabbit was injected at first with threshold doses (0.6 mℓ), and then with increased doses once, twice or three times every day (with a two-hour interval between repeated injections). After this, we checked the effect of doses known to be fatal. The results of the experiments are shown in Table 9 and graphically depicted in Fig. 55. As the table shows, as a result of /192 repeated systematic air injections begun with comparatively small doses, a clear increase takes place in the rabbits' resistance to the harmful effect of aeroembolism. In the very course of the experiments, after a number of injections of threshold doses (0.6 mℓ) of air, the rabbits acquire the ability to endure just as easily doses of 0.1, 1.5 and even 2 mℓ (cf. Table 9, rabbits 3-8). As a result, the animals turn out to be able to endure quantities of air that are known to be fatal. Thus, six rabbits

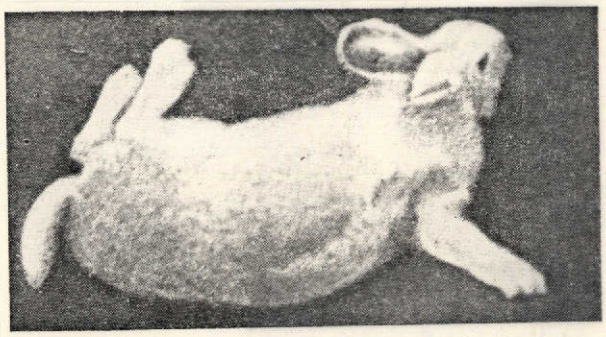

Fig. 53. Rabbit 8 min after intravenous injection of 1.5 mℓ air. Paralysis of rear part of body.

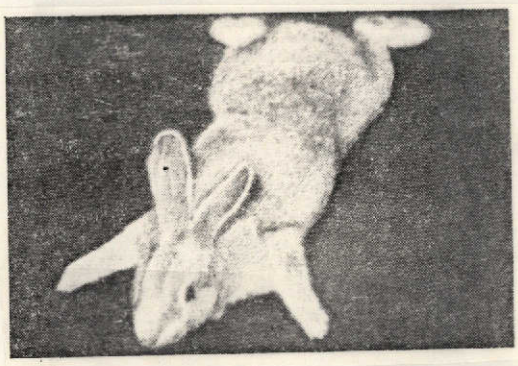

Fig. 54. Rabbit 2 min after intravenous injection of 3 mℓ air. Total paralysis, preagonal state.

endured the injection of 3.5-4 mℓ and more, and two of them (No. 6 and 7), 6.5 and 8 mℓ, i.e., doses more than four times in excess of the average fatal ones. These data point to the possibility of distinctively conditioning the organism to such an unusual effect as an intravenous injection of gases.

It should be noted that in undertaking such experiments, one might, it would seem, have feared accumulation phenomena, especially in those cases where each animal received three batches of air daily for a number of days (rabbits 10 and 11). But the experiments completely dispelled these misgivings and showed that with three daily injections of air, the increased resistance /193 proved to be no less pronounced.

Of interest are specially conducted experiments (18 experiments on five rabbits), in which we attempted to determine how rapidly gas bubbles injected into the blood are liquidated in the organism of rabbits that have not been conditioned to artificial aeroembolism and of those that have undergone such conditioning. To this end, at different times (ranging from 5 min to two hours after intravenously injecting the rabbits with easily endurable doses of air -- most often, 0.8 mℓ) we raised them in the pressure-chamber to an altitude of 4.5 km and kept them there for 5 min. At this altitude, intact rabbits display no substantial functional shifts apart from some general inhibition and slight panting. /194 During ascent and at the altitude, rabbits that had been injected beforehand with air developed severe functional disorders in the form of acute panting and dyspnea, opisthotonos, loss of equilibruim, pareses of the extremities and even spasms. These manifestations, which are undoubtedly connected with the spreading

TABLE 8. RESULTS OF INTRAVENOUSLY INJECTING RABBITS WITH DIFFERENT DOSES OF AIR IN CONTROL EXPERIMENTS

Animal number	Amount of injected air, ml	Reaction and outcome of experiment
1	9	General tonic spasms, death in 3rd minute
2	3	Paralysis of rear, then front extremities. Death in 3rd minute.
3	3	Same reactions. Death in 5th minute.
4	3	Very acute general depression, then paralysis of rear extremities, opisthotonos. Death in 16th minute.
5	2	Opisthotonos, general spasms, death in 3rd minute.
6	1.5	Same reaction. Death in 5th minute.
7	1.5	Steadily progressing panting, paralysis of rear extremities. Death in 17th minute.
8	1.5	Same reaction. Death in 12th minute.
9	1.8	Paralysis of rear extremities, clonic spasms of front part of body. Death in 4th minute.
10	1.3	Acute panting, opisthotonos, paralysis of rear extremities. Death in 6th minute.
11	1.3	Pareses of rear extremities. Survived.
12	1.3	Panting, general depression, opisthotonos. Survived.
13	1.0	Pronounced panting, general inhibition. Survived.
14	0.6	Panting, inconsiderable depression. Survived.
15	0.5	Same
16	0.3	Visible changes are absent.

TABLE 9. RESULTS OF INTRAVENOUSLY INJECTING AIR IN RABBITS AFTER CONDITIONING TO ARTIFICIAL AEROEMBOLISM

Animal number	Subliminal dose, mℓ	Duration of application, days	Doses injected after conditioning, mℓ	Reaction and outcome of experiment
1	0.6	6	2.5	Temporary general depression, transient paresis of rear extremities. Survived.
2	0.6	Same	2.5, then 4.0	Panting, temporary general depression. Survived.
3	0.6 0.1 1.5	6 4 5	2.0	Same
4	0.6 1.0 2.0	6 4 5	4.0	Very transient paresis of rear extremities. Survived.
5	1.0	5	2.0	Acute panting. Survived.
6	0.6 1.0 1.5	6 6 3	2.0, then 8.0	2.0 mℓ -- no persistent consequences; 0.8 mℓ -- flaccid paralysis of rear extremities. Survived.
7	0.6 0.8 1.5	6 (twice) Same (twice)	6.5 8.0	Transient pareses of rear extremities. Survived. General paralysis of body. Death.
8	0.6 1.0 1.3	5 6 5	3.0	Acute panting and general inhibition. Survived.
9	0.6	5(three times)	4.0	General paralysis of body. Death.
10	0.6 0.8	5(three times) 4(three times)	3.5	Transient paresis of rear extremities. Survived.
11	0.8 0.6	6(twice) 5(three times)	4.5	Temporary paralysis of rear extremities, panting. Survived.

of the intravenously injected air emboli during ascent to the altitude, were the more pronounced, the shorter the interval between air injection and ascent. When this interval was increased to 1 hour 30 min - 1 hour 45 min, they could no longer be detected. Rabbits raised to the altitude at such times after injection of 0.8 mℓ air, did not differ from intact animals. This gives grounds to conclude that for the indicated time, the organism manages to cope with the injected dose of air, and the air emboli at the moment of ascent turn out to be completely or mostly liquidated.

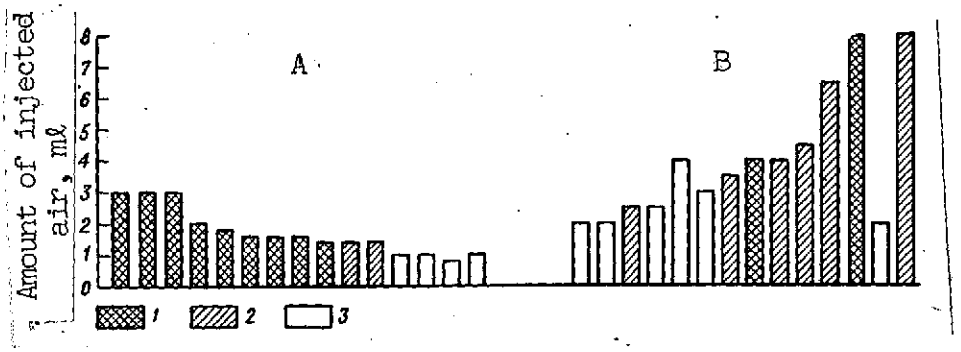

Fig. 55. Resistance to artificial aeroembolism in conditioned (B) and unconditioned (A) rabbits. 1 -- death; 2 -- transient functional disturbances; 3 -- absence of serious disturbances

It is very interesting that, after many days of conditioning, the same rabbits injected with air no longer displayed any functional disturbances during ascent to the altitude in the case of considerably shorter intervals (45 min - one hour) between air injection and ascent. Consequently, as a result of such conditioning, the organism acquires the ability to liquidate more quickly air emboli injected into the blood.

What attracts the attention about the obtained data, besides the basic fact of the organism's conditioning to artificial aeroembolism, is the preferential affection of the rear extremities and rear half of the body of the rabbits incident to injection of excessively large doses of air. Pareses and paralyses of the rear extremities and entire rear half of the body of the animals are the most typical symptoms of serious artificial aeroembolism. It should be recalled that these same symptoms are generally detected also in the case of serious decompression disorders. It is customary to explain caisson /195 pareses and paralyses of the lower half of man's body (the rear half, in the case of animals) either by gas formation in the

very tissue of the spinal cord, or by gas bubbles plugging up the vessels of the corresponding cerebral spinal segments.

For our cases, the first explanation is entirely out of the question; the second requires the assumption that emboli pass through the pulmonary capillaries and obstruct the cerebral spinal vessels from the side of the artery. But this, too, as our experiments showed, is out of the question. It remains for us to assume that the functions of the spinal cord suffer as a result of disturbances of blood circulation and simultaneously developing hypoxemia. It is possible that this explanation is more correct also for decompression parapareses and paraplegias, the more so, as these same manifestations are more regularly detected incident to serious degrees of general oxygen want, in particular incident to altitude hypoxemia.

Reactions of respiration and blood circulation incident to the conditioning of dogs to artificial and decompression aeroembolism

Jointly with V. I. Arsen'yeva and K. S. Yurova we conducted experiments on three dogs with their carotid arteries drawn out into a skin flap. The animals lay in a cradle and breathed through a mask; in the experiments, the pulse, respiration and pulmonary ventilation were graphically recorded and the electrocardiogram was taken.

Decompression effects consisted in keeping the animals at a depth of 45 m and rapid ascent to the surface. In the first experiments, the animals' stay amounted to 15 min and in subsequent experiments, it was increased each time by 5 min right up to the development of pronounced decompression disturbances in the animals (bends symptoms); after this, we set up an experiment with the foregoing stay reduced by 5 min; and subsequently resumed experiments involving its gradual increase, i.e., we did as was necessary in order to obtain the increased resistance effect. The intervals between experiments amounted to 2-3 days. Air was injected into the vein of the hock in amounts that increased from experiment to experiment (from 20-50 ml) at a rate of 1 ml every 2-2.5 sec. The physiological indices enumerated above were recorded immediately after the indicated effects (while the air was being injected) and, subsequently, every 10 min for an hour. If, in the experiments with decompression, the dog developed pronounced painful symptoms of the bends type (this was always noted in less than an hour), the recording of the physiological indices was continued for another 3-5 min, and then the animals were subjected to therapeutic recompression.

The purpose of the experiments was not confined to simply studying the reactions of respiration and blood circulation

during conditioning to decompression effects and artificial aeroembolism and to comparing the reactions in both of these cases. The experiments also had the purpose of looking into a question that is of importance as a matter of principle in the context of our investigation: does the above-mentioned promising effect of increased resistance takes place for decompression disturbances after repeated air injections and for artificial aeroembolism after repeated decompression effects? To this end, the order of the experiments was varied in an appropriate manner. In all, 187 experiments were conducted with decompression and 44 experiments with intravenous air injection. /196

The experiments showed that the changes in respiration and heart action in intact animals incident to decompression effects and intravenous air injection are very similar in character. In both cases, panting always develops with a more or less considerable increase in pulmonary ventilation and, as a rule, the pulse slows down. In the initial experiments, when the conditioning to the effects in question has not yet had time to reveal itself, the intensity of panting and bradycardia incident to decompression depends directly on the duration of the animal's exposure to pressure, and incident to air injection, on its dose and rate of administration. It is significant that the indicated changes in respiration and heart action incident to decompression arise not only in those experiments which terminate in development of the typical symptoms involving the extremities, but also in those which are not accompanied by these symptoms and, in view of the short duration of exposure "at the bottom", do not even threaten to develop.

Besides the general similarity between the reactions to decompression effects and to intravenous air injections, there are also certain differences between these reactions in the two cases. First of all, they concern the dynamics of the changes in respiration and heart action. Incident to artificial aeroembolism, panting and slowing down of the heartbeat are most pronounced in the first 5-10 min after air injection; then they gradually weaken and, in a number of cases, at the end of the experiment, after 50 minutes disappear entirely. But after decompression, panting and bradycardia develop gradually, attaining a maximum in experiments that are not accompanied by joint symptoms in the 20-30th minute, while in experiments where joint pains arise at a later time, they progress right up to their appearance. This circumstance becomes quite understandable, if we take into account that decompression-induced aeroembolic phenomena develop gradually, and at those depths and exposures which took place in the experiments, most often attain a maximum precisely 20-30 min after emergence from under pressure. The distinctive feature of the reactions in cases of artificial aeroembolism is short-lived tachycardia during the air injection, which is replaced immediately at the end of the injection by bradycardia.

But in cases of decompression effects, these reactions /197
assume a special character incident to development of symptoms involving the extremities. When such symptoms arise, bradycardia is replaced by an increase in the pulse rate, respiration becomes more frequent, but more superficial, and the dogs display a motor disturbance. All of this can obviously be explained by the onset of severe pains in the ailing extremity.

Fig. 56 shows the results of an experiment with a decompression effect that passed without the typical symptoms involving the extremities, but accompanied by the dog's pronounced general depression, which was noticeable even at the end of the recording when the animal was in a natural position. The figure shows increase in panting and development of bradycardia in the 20th minute after decompression. Fig. 57 shows a case with development of painful symptoms 38-40 min after decompression, when pronounced bradycardia was replaced by a relative increase in the pulse rate, while respiration became more frequent, but more superficial. Fig. 58 shows the results of an experiment with intravenous air injection. The oscillograms illustrate the tachycardia at the end of air injection, which was replaced by a slowing down of the pulse, and panting, which was most pronounced for a recording made 10 minutes after injection.

Figs. 59 and 60 show graphs reflecting the typical dynamics and magnitude of the functional shifts incident to artificial and decompression aeroembolism. It should be noted that the absence of substantial changes in the respiratory rate in experiments with decompression do not mean the absence of panting. In these cases, it developed mainly not as a change in rhythm, but as an increase in amplitude of respiratory movements.

The principal changes in the electrocardiograms in the case of decompression, as well as artificial, aeroembolism reduce to the following. Firstly, a shift of the heart's electrical axis toward the right is noted. This is borne out by the R_{III}/R_I index, which reflects the height ratio of the corresponding waves. In the control experiments, this index on the average equalled 1.7, with fluctuations for different experiments and different dogs ranging from 0.7 to 3.7. After decompression and, especially, after air injection, it generally increased considerably and, in a number of cases, proved to be equal to 5, 5.5 and even 6. It must be assumed that the dextrogram in these cases reflects an increased functional load on the right ventricle of the heart, which is connected with obstruction of the pulmonary capillaries with air emboli.

The second typical change in the ECG was a distortion of the T wave, which was particularly noticeable in the second lead. Being normally negative, the T_{II} wave incident to aeroembolism became positive or two-phase. This undoubtedly serious change

in the ECG is shown in Fig. 61, in which at the same time acute bradycardia can be easily seen, as well as arrhythmia, which arises in the present case after air injection.

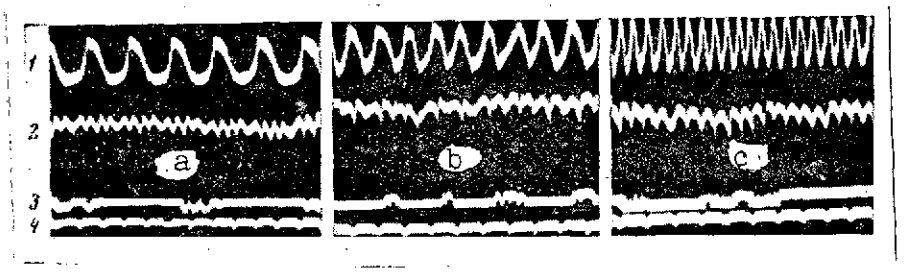

Fig. 56. Changes in respiration, pulse and pulmonary ventilation in Volchok 10 (a), 20 (b) and 30 (c) min after decompression from under a pressure of 4.5 psig (60-min exposure).
1 -- respiration; 2 -- pulse; 3 -- pulmonary ventilation (notch, 200 mℓ); 4 -- time indication, 1 sec.

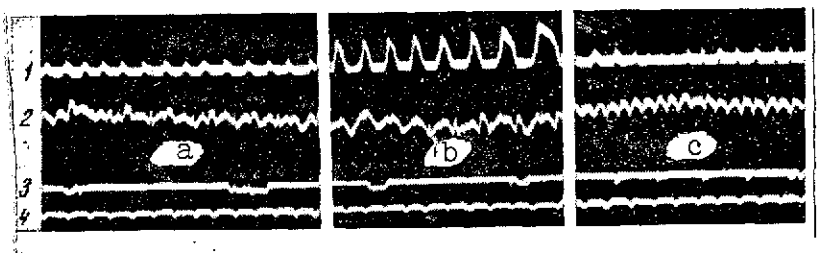

Fig. 57. Changes in respiration, pulse and pulmonary ventilation in Kudryash incident to development of typical decompression symptoms after a pressure effect of 4.5 psig (20-min exposure) 10 (a), 30 (b) and 40 (c) min after decompression. Rest of notation same as Fig. 56.

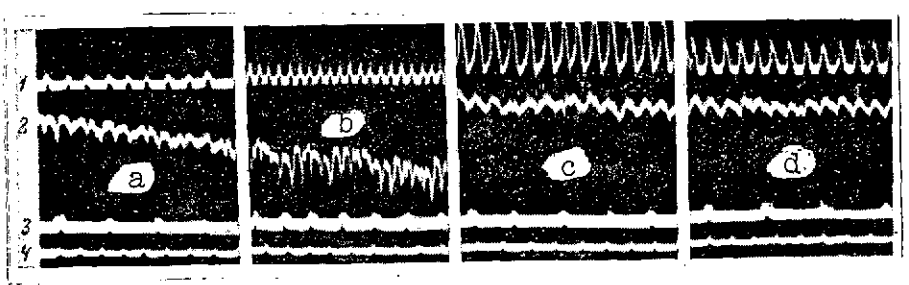

Fig. 58. Changes in respiration, pulse and pulmonary ventilation in Volchok incident to intravenous injection of 50 mℓ air for 2 min 15 sec. a -- before air injection; b -- at end of injection; c -- 10 min after injection; d -- 20 min after injection. Same notation as in Fig. 56.

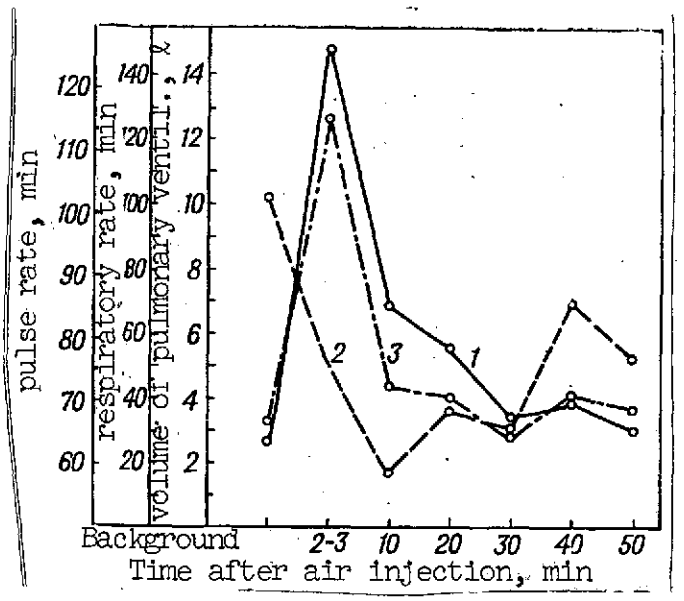

Fig. 59. Dynamics of change in pulse rate (2) respiration (1) and pulmonary ventilation (3) incident to intravenous injection of 50 ml air for 2 min 15 sec.

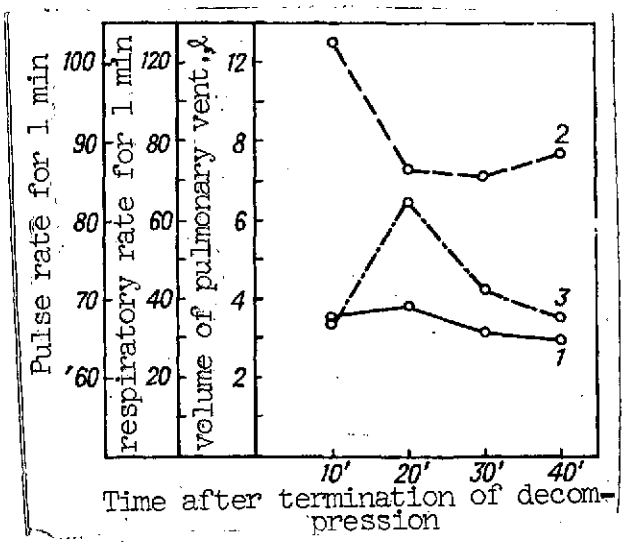

Fig. 60. Dynamics of change in pulse rate (2), respiration (1) and pulmonary ventilation (3) after decompression from under a pressure of 4.5 psig (50-min exposure) in Volchok.

In conformity with previous data, these experiments showed that incident to repeated effects the animals' resistance to intravenous air injection, as well as to decompression effects, increases considerably. The increase in resistance to artificial aeroembolism was expressed in the fact that after systematically repeated effects, the dogs endured with the previous ease an injection of twice as great a dose of air as compared with the initial injections. Thus, to judge from the outward appearance, general condition and rapidity of normalization of respiration and heart action, the dogs after repeated experiments with artificial aeroembolism endured an injection of 45-50 ml air as easily as they did at the beginning an injection of 20-25 ml. /200

The increase in the animals' resistance to decompression effects in the course of the experiments is borne out by the considerable increase in the safe staying time at a depth of 45 m that is determined by the onset of typical postdecompression symptoms involving the extremities. Thus, for Volchok this time increased from 40 to 60 min, though after a paresis of the rear extremity, which was not cured by recompression but disappeared in a month, it again dropped down to 50 min. For Timoshka, the safe time increased from 25 to 40 min, and for Kudryash from 15 to 40 min.

Most significant is the fact that the increase in the organism's resistance to the

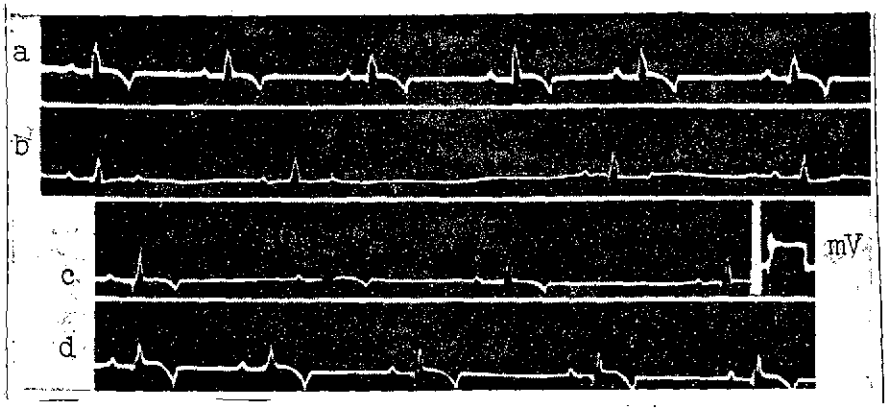

Fig. 61. Development of bradycardia and distortion of T wave of ECG (2nd lead) in Volchok incident to intravenous injection of 44 mℓ air for 50 sec. a -- before injection; 2 (b), 10 (c), and 20 (d) min after injection.

action of decompression was accompanied by a simultaneous increase in its resistance to artificial aeroembolism and vice versa. On one of the dogs (Kudryash), over a period of several months we carried out systematic experiments with the action of decompression and in the course of them, periodically checked the animal's reactions to injection of one and the same dose /201 (50 mℓ) of air. It turned out that in the course of time, these test injections of air are endured by the animals ever more easily, though no conditioning of the animal can be suspected in the present case: the air injections were separated by a period of two weeks.

The second dog (Volchok) was at first subjected to a repeated decompression effect; at the start of these experiments, its maximum permissible safe staying time on the bottom proved to be equal to 40 min, and at the end, 50 min. Then, in the course of a month, the animal was intravenously injected with air 22 times. After this, a check of the safe staying time under pressure showed that it had increased to 65 min. Consequently, conditioning to artificial aeroembolism even led to an additional increase in resistance, greater than that which was attained by repeated decompression effects (Fig. 62).

The observed crossed training effect leaves no doubt as to the fact that the increase in the organism's resistance to decompression effects incident to their repetition is based to a considerable extent on an improvement of the reaction evoked by aeroembolism.

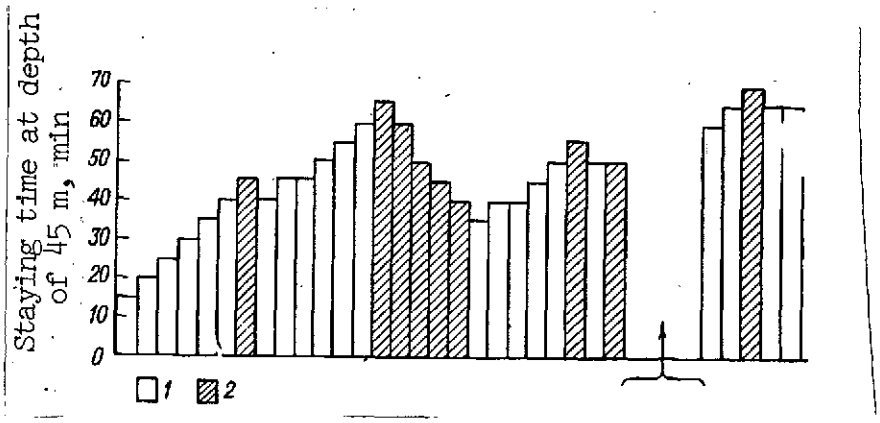

Fig. 62. Increase in resistance to decompression disturbances in Volchok after repeated intravenous air injections. 1 -- without decompression disorders; 2 -- with decompression disorders. The arrows indicate systematic intravenous air injections (22 times per month)

Actually, the experiments showed that as decompression effects and intravenous air injections are repeated, the reactions of respiration and of the cardiovascular system are somewhat modified. Bradycardia proves to be ever less pronounced, and in individual cases, does not arise at all and is even replaced by an increase in the heart rate. But panting persists, though it abates somewhat in intensity with repeated experiments. The above-mentioned distortion of the ECG waves decreases and is smoothed out, but the dextogram does not disappear. Finally, it is significant that as the experiments are repeated, panting /202 and tachycardia arise in the animals through a conditioned reflex from the experimental setup alone and the conditioned stimuli connected with aeroembolism (placing in the cradle, bubbling of the air through the hydraulic valve of the gas injection system, noise of the tape-drawing mechanism of the oscillograph). Thus, the organism's reactions to artificial and decompression aeroembolism improve in the first place in the sense that their components that are unfavorable to the organism disappear and defensive functional shifts are intensified and in the second place in the sense that these reactions, having become more full-fledged, are switched on earlier, even before the appearance of gas bubbles in the blood.

This last point deserves special attention. The switching on of defense reactions of respiration and blood circulation as a conditioned reflex incident to decompression effects may give rise to an effect that differs in principle from that which is obtained as a result of such reactions incident to the effect on the organism of different external factors. In the latter case, the advantage of the switching on of compensatory reactions

as a conditioned reflex consists in the fact that the organism prepares for battle with the harmful effect of the external factor even before it has acted; but by virtue of the factor that the organism is to run into, conditioned-reflex reactions can exert no effect.

In principle, the case is different with decompression effects. These effects influence the organism not in themselves, but only create those conditions under which the acting factor arises -- gas bubbles in the organism's internal environment. In this case, the conditioned-reflex defense reactions arising during decompression or immediately after it even before the appearance of gas bubbles, can altogether prevent or, at any rate, decrease their formation, owing to the fact that they reduce the organism's supersaturation with the inert gas. Consequently, incident to decompression effects, in contrast to other external effects, conditioned-reflex reactions not only prepare the organism beforehand for battle with the harmful factor, but can weaken it or even eliminate the possibility of its appearance.

It should be noted that under experimental as well as real conditions, very favorable conditions are created also for the conditioned-reflex switching on of compensatory reactions directed against decompression disturbances. The beginning of decompression is always clearly perceived by the organism: in the chamber, by noise and the change in temperature conditions; in the water, by the sensation of surfacing and in both cases, by the effect of the drop in pressure on the ear and larynx. But the possibility for bubbles to appear in the organism is created only after a certain, often short, time after the beginning of decompression. During this time, conditioned-reflex reactions, the signal for whose triggering was the beginning of decompression, can reveal to the fullest extent their great positive role. /203

But for man, with his second signaling system, the possibilities for early conditioned-reflex switching on of defense reactions incident to decompression effects acquire a still wider scope. Man's estimate of the staying time under pressure, his knowledge of the deadline for the command involving impending decompression (get ready to surface) and the different actions that he perceives must be performed before decompression, can serve as a signal for the switching on of defense reactions that prevent the development of caisson phenomena -- provided, of course, that the organism has already encountered them earlier.

Thus, it must be assumed that a very great role in the increase in the organism's resistance to decompression disturbances is played by the conditioned-reflex mechanism underlying the switching on of defense reactions.

Gaseous composition of the arterial blood incident to artificial and decompression aeroembolism

Proceeding from the indubitable fact that in the case of pronounced caisson phenomena, the pulmonary capillaries are plugged up with gas bubbles on a mass scale, it can already be assumed a priori that pulmonary gas exchange and oxygenization of the blood will be disturbed at the same time. This is confirmed by individual unsystematic data in the literature of former and recent times [Hoppe-Seyler, 1857; Bert, 1978; Behnke, 1951]. Still open, however, is the question whether pulmonary gas exchange is disturbed only incident to pronounced decompression disorders, or whether the changes in it and, correspondingly, in the gaseous composition of the arterial blood begin earlier and accompany also mild cases of caisson phenomena that are unattended by the development of specific decompression symptoms of the bends type. To elucidate this question, jointly with A. A. Savich we conducted 12 chronic experiments on four dogs aimed at studying the gaseous composition of the blood incident to such decompression effects as caused pronounced bends symptoms and such as led to no visible functional disturbances (Table 10). On six dogs, moreover, we conducted crucial experiments whose purpose was to compare, during the development of artificial and decompression aeroembolism, the changes in the gaseous composition of the arterial blood with the intensity of the aeroembolic phenomena and the reactions of the respiration and blood circulation.

In the chronic experiments, the oxygen content of the blood before the effect varied in most of the dogs within physiological limits. An exception was the dog Druzhok, in which the oxygen content was somewhat lower than normal. The carbon dioxide concentration in the arterial blood of most of the dogs before the /204
effect was lower than usual, which was apparently connected with the fact that the dogs, in spite of preconditioning to all of the experimental conditions (including pricking of the skin and puncture of the artery), nevertheless reacted to this painful stimulation -- they developed panting of the tachypnea type. An exception was the same dog Druzhok; the quantity of carbon dioxide in its blood corresponded to normal values.

In five experiments, the dogs, after staying under pressure and undergoing decompression, developed pronounced bends symptoms; in two cases, they were distinguished by their particular intensity. In seven experiments, these symptoms were absent, and the dogs' general condition underwent no significant changes. The matter was confined to a moderate increase in the respiratory rate and, in some dogs, in considerable general inhibition.

The oxygen content in the arterial blood of the animals after the effects proved to be more or less reduced in all

TABLE 10. CHANGE IN GASEOUS COMPOSITION OF THE ARTERIAL BLOOD IN DOGS INDICENT TO THE DEVELOPMENT OF DECOMPRESSION PHENOMENA

Dog	Depth, m	Exposure, min	Time after decompression, min	Appearance of symptoms of the bends type	Blood gases, vol.% Background O_2	Background CO_2	After effect O_2	After effect CO_2
Sedoy	45	50	10	Pronounced	22,05	39,93	21,56	41,26
Zor'ka	45	50	10	"	24,09	41,20	23,85	39,32
Druzhok	45	40	17	Very severe	15,00	44,96	7,86	52,43
	45	35	19	None	16,42	44,96	13,48	46,05
Mishka	45	45	19	Pronounced	21,81	35,40	15,93	43,90
	45	45	10	Very severe	22,21	39,81	13,57	45,46
Sedoy	16	240	80	None	21,47	37,39	21,71	39,27
Zor'ka	24	240	23	" *	23,43	37,39	21,47	39,81
Mishka	16	240	23	"	19,74	38,09	15,25	39,27
	45	40	25	"	15,71	44,92	13,82	47,08
			60	"			11,82	46,54
			101	"			20,48	41,43
Druzhok	20	300	35	"	15,8	44,7	13,86	45,54
			60	"			18,87	39,51
Sedoy	20	240	30	"	21,9	38,6	20,05	39,20

* Subsequent ascent to an altitude revealed the presence of "latent" bubbles.

experiments with the exception of one, in which the blood sample was taken a long time (80 min) after decompression. The reduction in the oxygen content was especially sharp in three experiments involving development of pronounced bends symptoms (by 5.6, 7.5 and even 8.5 vol.%); it is noteworthy that these three experiments include two in which the bends symptoms were also particularly sharp. In two cases involving development of the symptoms, the drop in the oxygen content was very small; such a result may perhaps be due to the fact that the dogs in these experiments reacted very violently to the development of painful symptoms and developed very intense hyperpnea. /205

In the experiments in which bends symptoms were absent, the reduction in the oxygen content of the arterial blood (the first blood samples) was also very considerable (approximately by 2.3 and 4.5 vol. %). In all cases but one, the carbon dioxide content in the dogs' arterial blood after the decompression effect was increased as compared with its initial value.[15] The

[15] In most cases, the absolute figures stayed below the normal ones, the reason for this, as has already been pointed out, clearly being the tachypnea that arose incident to taking the blood sample.

case in which it dropped was one of the experiments with violent hyperpnea caused by painful symptoms.

In the experiments involving development of pronounced bends symptoms (with the exception of those just mentioned), the quantity of carbon dioxide in the arterial blood after decompression increased very sharply (in three cases, by 5.65, 7.47 and even 8.5 vol. %). It is conceivable that this was in part connected with the animals' general motor reactions that accompanied the development of painful decompression symptoms. But the main reason for such a great rise in the CO_2 content was undoubtedly postdecompression aeroembolism inasmuch as it is known that even incident to the most intensive muscular work, the quantity of carbon dioxide in the arterial blood never increases to such an extent.

In the experiments involving no bends systems, the carbon dioxide content in the arterial blood increased in two cases by a small amount (0.6 and 0.84 vol. %); but in the remaining five experiments, it increased considerably (by 1.09-2.42 vol.%).

Noteworthy are two experiments in which, after the decompression effect, arterial blood samples were taken repeatedly. One of these experiments shows that in spite of the absence of decompression disturbances (bends symptoms), the oxygen content may sometimes drop, while the carbon dioxide content remains increased for a whole hour after decompression.

Most experiments point to the fact that a longer or shorter time after decompression, when it is clear that the aeroembolic phenomena have already passed in their entirety, the gaseous composition of the arterial blood not only becomes normalized, but undergoes shifts that are opposite to those which were caused by the decompression effect, i.e., the blood's oxygen content turned out to be higher and the carbon dioxide content, lower /206 than before the dog was placed in the chamber.

The main result of the chronic experiments was that they showed the presence of clear and regular shifts in the gaseous composition of the arterial blood not only in cases of manifest decompression disturbances (which, as has been mentioned, a number of authors have already pointed out), but also in those cases from which specific decompression symptoms are absent, the organism's condition is practically quite normal and the development of caisson phenomena in the organism is outwardly expressed only in some slight increase in the respiratory rate.

The crucial experiments showed that incident to decompression aeroembolism, as well as incident to intravenous air injection, the very same shifts that we have just described take place in the gas composition of the arterial blood: reduction in its

oxygen content and increase in its carbon dioxide content. At the same time, it was plain to see that there was a direct dependence among intensity of aeroembolic phenomena, shifts in the gaseous composition of the arterial blood and changes in respiration and cardiovascular activity.

In the case of decompression aeroembolism, when small amounts of gas are first being formed in the veins, an inconsiderable decrease in the oxygen and increase in the carbon dioxide take place in the arterial blood, and slight panting and not very pronounced shifts in cardiovascular activity can be observed. As the caisson phenomena develop and the gas formation intensifies, the shifts in the gaseous composition of the arterial blood progress, in which they are matched by the intensification of the reactions of the respiratory and cardiovascular systems.

In the case of artificial aeroembolism, the extent of the changes in the gaseous composition of the arterial blood, as well as the degree of manifestation of the reactions of the respiratory and cardiovascular systems, depend directly on the amount of air injected into the vein (Fig. 63).

The direct and constant dependence among intensity of artificial or decompression aeroembolism, extent of changes in the gaseous composition of the arterial blood and degree of manifestation of the reactions of the respiration and blood circulation point to the fact that these phenomena stand in a causal relationship with one another.

The experiments described in the present chapter showed directly that, firstly, the organism can be conditioned to aeroembolism and, secondly, that this conditioning is the basis for the increase in resistance to decompression disturbances incident to repeated decompression effects of gradually increasing intensity. Conditioning to aeroembolism and increases in resistance to decompression disturbances are ensured, as is clear from the foregoing material, by reactions of the respiration and blood circulation. In accordance with a principle that is common to all conditioning, these reactions incident to repeated decompression effects or repeated air injections become ever more perfected with the passage of time. Their pathological components smoothen out and even disappear, and their components directed at reducing and liquidating those physiological and biochemical shifts which arise as a result of the aeroembolic process, intensify. Such perfecting of reactions is characteristic of the organism's adaptation to the effect of the most diverse external factors and the effect of the most diverse changes in conditions. Consequently, decompression aeroembolism in this respect constitutes no exception. /207

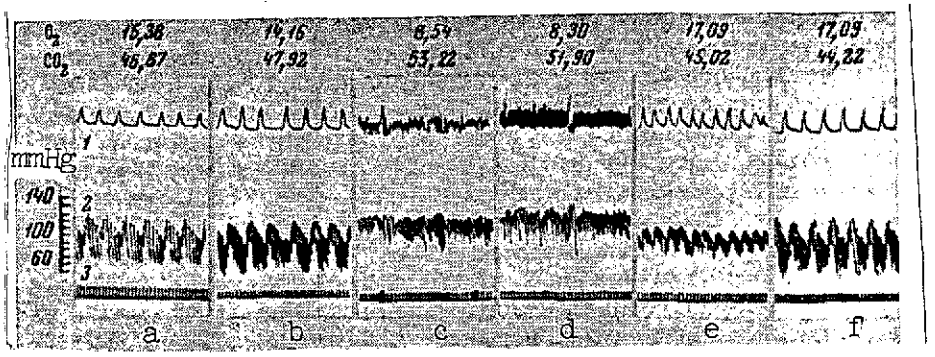

Fig. 63. Changes in respiration, blood pressure and O_2 and CO_2 content (vol.%) in the arterial blood of an anesthetized dog incident to intravenous air injection. a -- background; b -- 10 min after injecting 50 cm^3 air for 30 sec; c -- 2 min after additional injection of 200 cm^3 air for 2 min 30 sec; 2 (d), 17 (e) and 42 (f) minutes after additional injection of 150 cm^3 air for 1 min. 1 -- respiration; 2 -- arterial blood pressure; 3 -- time indication, 1 sec.

The experiments we carried out permit us to draw a quite definite conclusion about the mechanism governing the development of defense reactions incident to caisson phenomena. Clearly, the main immediate cause of these general defense reactions that manifest themselves in intensified respiration, pulmonary ventilation, heart action (especially of the right ventricle) and blood circulation are the shifts in the gaseous composition of the arterial blood that arise as a result of aeroembolism of the pulmonary capillaries. These shifts, as experiments have shown, already arise incident to the mildest forms of caisson phenomena, when no specific decompression symptoms or functional disturbances are present, and researchers, not noticing the initial reactions of the respiration and cardiovascular activity, often do not even suspect that gas formation is already taking place in the organism. Moreover, this early stage in the development of caisson phenomena and the mentioned initial compensatory reactions of the respiration and blood circulation that are /208 caused by the change in gaseous composition of the arterial blood, to a considerable extent predetermine the entire subsequent course of the matter, as well as the possibility that pronounced decompression disturbances may arise.

The shifts in the gaseous composition of the arterial blood concern oxygen as well as carbon dioxide, the reduction in the O_2 content and increase in the CO_2 content in the arterial blood

generally corresponding in most cases to one another. Consequently, we can say that the triggering mechanism of defense reactions in response to development of the aeroembolic process is an asphyxial factor. In a number of cases, the changes in the quantitative oxygen and carbon dioxide contents that we observed in the arterial blood were small and may be of little physiological significance.

Actually, under ordinary circumstances, such fluctuations in the O_2 or CO_2 contents, when they are connected with changes in the blood's oxygen and carbon dioxide capacity, are of no great importance. But in our case, the shifts in the oxygen and carbon dioxide contents are caused by a change in the pressure of these gases in the arterial blood, which is regulated by the organism with an accuracy to millimeters of mercury. Hence, it follows that under the given conditions, it is impossible to leave out of account even the slightest variations in the amounts of O_2 and CO_2 in the arterial blood. We possess no direct data on whether the reduction in the oxygen or carbon dioxide pressure incident to development of caisson phenomena is of great significance. It seems, however, that the role of arterial hypercapnia must be no less, and perhaps even greater, than that of hypoxemia inasmuch as CO_2, which is a specific stimulant of the respiratory and cardiovascular centers, at the same time exerts a pronounced local vasodilatory effect, which is very important for the gas formation process in the capillaries and for the entire course of caisson phenomena.

Be that as it may, the shifts in the gaseous composition of the arterial blood must be regarded as a fundamental and inherent link in the organism's general reactions to postdecompression aeroembolism. But it would be a mistake to reduce the mechanism of the organism's reactions in response to aeroembolism to no more than changes in the gaseous composition of the blood. There can be no doubt that a number of other reflex mechanisms also play a role in the changes in respiration and, especially, blood circulation incident to aeroembolism. These are, above all, various reflexes triggered by a change in hemodynamics and ensuring self-regulation of the blood circulation. Plugging up of the pulmonary capillaries leads to a rise in the blood pressure of the pulmonary arteries.

As a result of this, as was shown by V. V. Parin [1934], and then Schwiegk [1935], the vessels of the greater circulatory system are involved in a reflex directed at reducing the total arterial pressure. The belaboring of the blood flow through embolized pulmonary vessels must lead to overfilling of the venous blood of the venae cavae and their distention; this, when the blood is supersaturated with a considerable amount of nitrogen, is probably furthered also by purely physiological

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circumstances -- intensive gas formation in the venous blood and the concomitant increase in the total volume of blood and gas filling the venous system. This may give rise to a reflex that is very useful under the circumstances: the Bainbridge reflex (increase in heart rate) directed at easing the venous system and intensifying the pumping of the blood through the lesser circulatory system.

The drop in blood pressure in the aorta and its ramifications arising as a very close direct consequence of aeroembolism of the pulmonary vessels must trigger powerful cardiovascular reflexes from the arch of the aorta and the carotid sinus. In this case, the cardiac component of the reflex in question (increase in heart rate) stands out as being absolutely useful; but the vascular component (stenosis of the arterioles of the greater circulatory system), which is useful for the general hemodynamics inasmuch as it causes a rise in arterial pressure, may lead to considerable local damage; in those organs where the arterioles contract, the capillary blood flow is slowed down and conditions are created which are conducive to gas formation in the blood and, under certain conditions, in the tissues, too.

The reflexes evoked by general changes in the hemodynamics and, in particular, in the total arterial pressure, concern not only the cardiovascular system, but also the respiration; in particular, the drop in arterial pressure, acting on the baro-centers of the carotid sinus, may cause a reflex intensification of respiration. Moreover, the simultaneous involvement of the respiration and cardiovascular system in the reaction may, under the conditions in question as well as in other cases, be determined by very close internal interrelations between the specific centers.

It must not be forgotten that, besides changes in the gaseous composition of the blood and hemodynamic shifts in the forming of general respiratory and circulatory reactions, decompression aeroembolism can be influenced by reflexes evoked by the purely mechanical effect that gas bubbles arriving in the lungs exert on the capillary walls and surrounding pulmonary tissue. Clearly, it is by precisely this effect of air emboli that we must explain the decompression disturbances often encountered in diving practice and, especially, incident to rarefaction of the atmosphere in the form of pains below the chest and an uncontrollable dry cough, which are usually accompanied by abundant cold sweat with a simultaneous deterioration of the organism's general condition.

There can be no doubt that the mechanical effect of gas bubbles on vascular walls plays a very important role in local reactions to aeroembolic phenomena, which were already discussed

in detail by V. V. Pashutin [1881]. These local reactions can also have a twofold significance: angiospasm of the arterioles will always have a negative significance here and dilatation, a positive one. Clearly, incident to local disturbances of the blood flow, when products of the metabolism come forward as the stimuli, the initial contraction of the vessels may be replaced by their active distention.

In investigations on human beings (the results of which will be discussed separately), we generally noted, when decompression itching developed at an altitude, a significant rise in temperature of the itching sections of skin, which undoubtedly testified to the increased intensity of the blood flow in the corresponding regions. This fact cannot be interpreted otherwise than as a local vascular defense reaction caused by gas bubbles that have formed in the blood and directed at liquidating local disturbances of the blood circulation that are about to take place.

Nor, finally, when the organism is supersaturated with great amounts of an inert gas, must we leave out of account the possibility of intratissue formation of gas bubbles that can have the most diverse consequences, depending on their size and location. But in speaking of extravascular postdecompression gas formation, it is always necessary to bear in mind two circumstances that are important as a matter of principle: firstly, that gas bubbles are formed in the tissues, as has been shown by A. P. Brestkin, at greater degrees of supersaturation than in the blood, and, secondly, that the possibility of intratissue gas formation depends first of all on the state of their blood supply, as well as on the blood circulation as a whole and on respiration.

Thus, the organism's general reaction and, above all, the general reaction of respiration and blood circulation incident to development of caisson phenomena in the organism proves to be very complicated; it includes a number of reflexes, some of which have a positive significance, others a twofold significance and still others, a negative significance. At the same time, as ensues from the experimental material, the dominant role belongs to the general reactions of the cardiovascular system and respiration that arise as a result of arterial hypoxemia and hypercapnia and have a pronounced defensive character. It is these reactions that are the basis for the organism's adaptation and increased resistance to decompression disturbances.

The factual material presented in this chapter can be summarized in the following manner.

Incident to repeated systematic intravenous air injections, the resistance of animals to artificial aeroembolism increases considerably.

Conditioning the organism to artificial aeroembolism increases its resistance to decompression disturbances, just as conditioning to decompression effects increases its resistance to intravenous injection of gases.

The organism's increased resistance to decompression dis- /211
turbances incident to repeated decompression effects is based on a perfecting of the defense reactions of the respiratory and cardiovascular systems that are evoked by aeroembolism. Of considerable significance here is the conditioned-reflex triggering of these reactions.

A leading role in the mechanism governing the development of these reactions is played by the changes in gaseous composition of the arterial blood (hypoxemia and hypercapnia) that arise because of aeroembolism of the pulmonary capillaries. These changes already arise incident to mild forms of caisson phenomena that are not accompanied by visible functional disturbances.

CHAPTER 11

PROPHYLAXIS AND THERAPY OF DECOMPRESSION DISORDERS

Study of the conditions and mechanism of development of the decompression disturbances that arise after a stay under increased pressure, however great their theoretical and general physiological interest may be, must not be regarded as an end in itself -- its ultimate vital purpose is to perfect the prophylaxis of decompression disorders and to ensure the safety and increase the efficiency of divers and caisson workers.

The first question to be raised by the obtained data was whether it might not be possible, with the aid of some additional effects on the organism, to enhance those compensatory defense reactions which are the basis for its active opposition to decompression phenomena and determine its resistance to decompression disorders. Inasmuch as experiments have shown that these reactions generally reduce to intensification of the respiration and blood circulation, it was natural to investigate the effect of pharmacological stimulation of the given functions during the organism's desaturation from an inert gas, i.e., during and after decompression. To this end, we set up heuristic experiments in which we tested the effect of stimulants of cardiovascular activity -- caffeine and adrenaline -- and respiratory stimulants -- lobeline, Korkoniy and Antifein.

Further, the obtained material gave us cause to return to a question raised long ago by practice: what is the effect of muscular work on the onset and development of decompression

disturbances? One side of this question -- the significance of a physical load during the action of increased pressure -- has /212 been quite clear for a long time: muscular work on the sea bottom, by intensifying the organism's saturation with the inert gas, significantly increases the danger of decompression disturbances developing after return from the depth. As far as the second side of the question is concerned -- the role of a physical load during the organism's desaturation from the inert gas incident to decompression from under increased pressure --, no definite, scientifically well-founded opinion exists down to the present time. From the data of altitude physiology, as we shall see below, it ensues with certitude that muscular work incident to decompression effects connected with rarefaction of the atmosphere, plays a negative role, i.e., it is conducive to the development of decompression disorders, but no less certain is the fact that muscular activity is a powerful natural respiratory and circulatory stimulant, which, it would seem, in the stage of desaturation of the organism from the inert gas, must have a direct positive significance. In this connection, we undertook experiments on dogs involving a muscular load after decompression.

Finally, experiments were conducted concerning another method of decompression disturbance prophylaxis, namely: setting up such conditions in the process of diving as would in themselves, independently of the organism's defense reactions, reduce the danger of decompression disorders developing without at the same time lengthening decompression time. This task reduces to utilization of different gaseous mixtures in the process of diving and can in principle be solved in two directions.

Firstly, incident to diving, gaseous mixtures can be used with an increased, but still safe from the standpoint of toxic effect, oxygen content and a correspondingly decreased content of the inert gas. In this direction, very appreciable progress has already been achieved. V. L. Zal'tsman and his coworkers [1961] have developed air-oxygen and helium-nitrogen-oxygen mixtures permitting divers' safe decompression times to be sharply reduced.

Secondly, an alternating supply of gaseous mixtures of different compositions can be used. Yunkin's study [1965] showed that promising from the standpoint of decompression disturbance prophylaxis and reduction of decompression time may be the alternate use of oxygen-poor and oxygen-rich gaseous mixtures. But the question does not reduce merely to a change in the oxygen content of the inspired mixture. At stake may be the possible significance of alternating the inert gases inspired by a man incident to diving.

As early as 1941, N. V. Lazarev in his monograph "Biological

effects of gases under pressure" advanced the idea of alternate inspiration of gaseous mixtures of different compositions under increased pressure conditions for the purpose of preventing decompression disorders. The basis for this idea was the well- /213
known Dalton-Henry law, according to which the diffusion of any gas from one medium into another takes place independently of the content and the concentration of other gases in these media. From this it should have followed that when gaseous mixtures containing different inert gases are inspired alternately, each of these gases will now penetrate into the organism, now (incident to alternation of the mixtures) diffuse in the opposite direction and escape into the external environment. Under such conditions, no supersaturation of the organism with inert gases should set in, and, consequently, the danger of decompression disorders would be removed. Lazarus did not subject his hypothesis to experimental verification, but somewhat later he, jointly with Kurbatov, undertook a detailed mathematical analysis of his idea, as a result of which the authors arrived at the conclusion that it was untenable. The analysis indicated that when inert gases are alternated, the organism's total supersaturation with them will not decrease, and, consequently, the danger of decompression disorders developing will not decrease, either.

Theoretical considerations fully justified this conclusion, which, however, did not, as the authors themselves pointed out, obviate the necessity of checking it experimentally. Inasmuch as no such check has been carried out down to the present time, we undertook the appropriate experiments involving alternation of the inert gases (nitrogen and helium) breathed by animals during their stay at the sea bottom.

It should be noted that even with a negative result, which was to be expected here on theoretical grounds, the question of the value of using different inert gases for decompression disorder prophylaxis could not yet have been regarded as definitely solved.

It was necessary to check still another version of using different gases: not their rhythmical alternation during the stay at the bottom, but the replacement of one gas by another in different diving and decompression stages. With such a version, it is possible to allow for the different diffusibilities of different gases and, consequently, the different rates of saturation of the organism with these gases and desaturation from them. It seemed probable that, by taking into account these properties of gases and using different gases in different stages of diving, depending on the latter's concrete conditions, one could derive a certain benefit from the standpoint of preventing decompression disorders. It must be mentioned that our investigations in this direction, which will shortly be the subject

of discussion, belong to the time when the Swiss physicist Keller was causing a sensation with his diving exploits, which were breaking all records for brevity of surfacing from the deepest permissible depths. On the basis of what was then known about these achievements, it could be supposed that they /214 were based on rational interchange of gaseous mixtures of different compositions in the process of submerging and surfacing. But Keller's secret was still being kept at that time, and the question required experimental study. To obtain rough data, we undertook a separate series of experiments.

It should be noted that the experimental working out of methods of decompression disturbance prophylaxis and, even more so, therapy, was not the specific purpose of our study. Accordingly, our experiments involving study of the above-mentioned questions had a heuristic character.

They were undertaken for the purpose of evaluating ways and means of perfecting decompression disturbance prophylaxis on the basis of physiological analysis of the mechanism governing the development of decompression disorders.

Effect of respiratory and cardiovascular stimulants on the genesis of decompression disorders

We studied the effect of caffeine and and lobeline mixtures on the genesis of decompression disorders. One dog was kept for four hours under a pressure of 2.6 psig, which corresponded to the established coefficient and under a pressure of 2.8 psig, when this coefficient turned out to be excessive. A second dog was kept under a pressure of 4 psig for 40 min, after which rapid single-step decompression to the surface was also effected.

Control experiments in which the animals were not subjected to any additional effects alternated with experiments in which caffeine and lobeline were used. Ten min before the end of the increased pressure effect, the experimenter entered the chamber through the airlock and injected the dogs subcutaneously with mixtures of 3.0 ml caffeine solution and 1 ml lobeline solution (the dogs weighed 18 and 19 kg); the experimenter then came out through the antechamber with one or two short halts, and on expiration of the animals' staying time under pressure, they were subjected to rapid decompression. The lobeline and caffeine injections in the indicated doses stimulated the dogs' respiration moderately and increased their pulse rate by 12-20/min.

The results of the experiments are shown in Fig. 64. They point to the favorable effect of caffeine and lobeline: in two out of three control experiments, the dog Druzhok developed

pronounced decompression disorders (bends symptoms and corresponding change in general condition), whereas two experiments using caffeine and lobeline elapsed with no functional disturbances; in the case of the dog Sokol, all three control experiments were accompanied by decompression disorders, while out of three experiments with caffeine and lobeline, these disorders developed only in one. /215

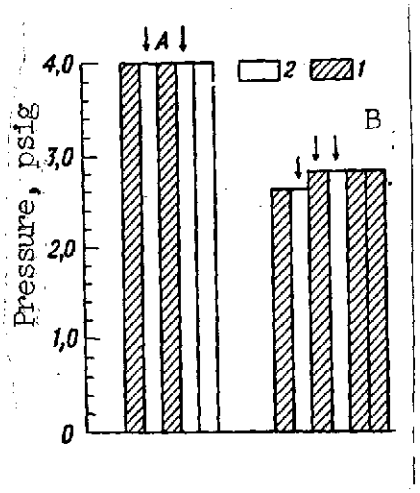

Fig. 64. Effect of caffeine and lobeline on development of decompression disorders in Druzhok (A) after a stay under a pressure of 4 psig for 40 min, and in Sokol (B) after a stay under a pressure of 2.6-2.8 psig for four hours. Arrows point to experiments in which drugs were injected; 1 -- with disturbances; 2 -- without disturbances.

In general, if we add up the results obtained for both dogs, decompression disturbances developed in five out of six control experiments, while they developed in only one out of five experiments using caffeine and lobeline. These experiments do not suffice for categorical conclusions, but on their basis we are entitled to suppose that a mixture of caffeine and lobeline used before decompression decreases the likelihood that decompression disorders will develop.

In three dogs, we studied the effect of Korkoniy and Antifein on the genesis of decompression disorders. These powerful respiratory stimulants, which were studied in detail in M. Ya. Mikhel'son's laboratory, when injected subcutaneously in doses of 0.8-1.0 mg/1 kg body weight, have a very pronounced hyperventilation effect lasting several tens of minutes. It was natural to expect that injecting them while the organism was being desaturated from nitrogen would accelerate its liberation and reduce the danger of decompression disturbances developing. In the experiments, after being kept at a depth of 45 or 60 m, the dogs were subjected to two-step decompression with one intermediate halt; at this halt ranging in depth from 12 to 21 m, the dogs were injected with Korkoniy or Antifein. The lengthy two-step decompression must have created better conditions for the indicated substances to affect desaturation as compared with single-stepped decompression.

The dogs' staying time at a depth of 45 m varied from 45 to 75 min, and at a depth of 60 m, from 30 to 50 min; the staying time at the intermediate halt varied from 30 to 70 min. These times varied as a function of the dogs' resistance to decompression disturbances and were selected so that decompression disorders would arise in approximately half of the control experiments. The dogs were injected with Korkoniy or Antifein in doses of 0.8-1.0 mg/kg in the first few minutes of their /216
stay at the intermediate halt. Four-six min after injection, the dogs started panting; the panting intensified under our eyes and rapidly attained a very high intensity, and the frequency as well as depth of respiratory movements increased by several factors. The hyperventilation effect reached a maximum 10-12 min after injection; in 18-20 min it began gradually to weaken, but disappeared definitely only after a lapse of 30-35 min.

TABLE 11. RESULTS OF EXPERIMENTS USING KORKONIY AND ANTIFEIN

Dog	Control			Korkoniy			Antifein		
	In all	Without disturbances	With disturbances	In all	Without disturbances	With disturbances	In all	Without disturbances	With disturbances
Sedoy	7	4	3	7	2	5	4	1	3
Bogatyr'	7	3	4	6	2	4	2	1	1
Sokol	7	4	3	7	3	4	2	1	1
Total	21	11	10	20	7	11	8	3	5

In spite of the extremely intensive and long-lasting hyperventilation of the lungs, which, it would seem, must have accelerated considerably the organism's desaturation from nitrogen and prevented the development of decompression disorders, neither Korkoniy nor Antifein yielded any positive effect. What is more, in experiments using them, decompression disturbances (bends symptoms) developed even more frequently, though in a number of cases more mildly, than in the control experiments (Table 11). Such a result was unexpected and had to be explained. Inasmuch as the organism's desaturation from nitrogen depends not only on the respiration, but also on the blood circulation, a natural explanation was that the latter changes under the effect of Korkoniy and Antifein; the more so, as in the studies

conducted in M. Ya. Mikel'son's laboratory, devoted to the pharmacological properties of these substances, this question was not studied. On four dogs, we undertook crucial experiments in which we recorded the respiration, volume of pulmonary ventilation and arterial blood pressure; simultaneously with this direct method, with the aid of Ludwig's stromuhr we determined the volumetric flow rate of the blood in one of the carotid arteries.

In these experiments, we studied the dynamics of the changes of all of the enumerated indices incident to intravenous injection of Korkoniy and Antifein in doses of 1.0 mg/1 kg body weight. The experiments showed that, simultaneously with intense stimulation of the respiration and amplification of /218 pulmonary ventilation, Korkoniy and Antifein sharply decreased the general intensity of the blood circulation (Fig. 65). Before the Korkoniy injection, the dog's pulmonary ventilation was equal to 8.5 ℓ/min and the volumetric flow rate of the blood in the carotid artery amounted to 150 mℓ every 1 min 16 sec. After the Korkoniy injection, which caused panting and a sharp, short-lived pressor effect with a subsequent drop in the blood pressure and its stabilization at a lower level, pulmonary ventilation rose to 18.5 ℓ/min, while the volumetric flow rate of the blood dropped no less sharply: the time of passage of the same volume of blood -- 150 mℓ -- through the clock increased to 2 min 42 sec. A more than twofold increase in pulmonary ventilation accompanied a more than twofold slowing down of the blood flow. The results of all such tests in the experiments on all four dogs are shown in Fig. 66. They show that the described effect is strictly regular and that the extent of slowing down of the blood flow is directly dependent on the extent of increase in pulmonary ventilation.

These data completely explain the absence of a positive effect of Korkoniy and Antifein in the decompression experiments: the intensification of pulmonary ventilation with a simultaneous sharp slowing down of the blood circulation could not accelerate desaturation or prevent the development of caisson phenomena. On the other hand, the slowing down of the blood flow, by promoting intensified gas formation in the organism, worsened the matter.

The observed divergences between respiratory and circulatory shifts incident to the action of Korkoniy and Antifein are probably due to maintenance of normal carbon dioxide pressure and content in the organism. A considerable increase in pulmonary ventilation with unchanged intensity of blood circulation would inevitably lead to acute hypocapnia and functional disorders. Incident to artificial stimulation of the respiration, a slowing down of the blood flow is the only way of retaining carbon dioxide in the organism and obviously arises as a reaction directed at

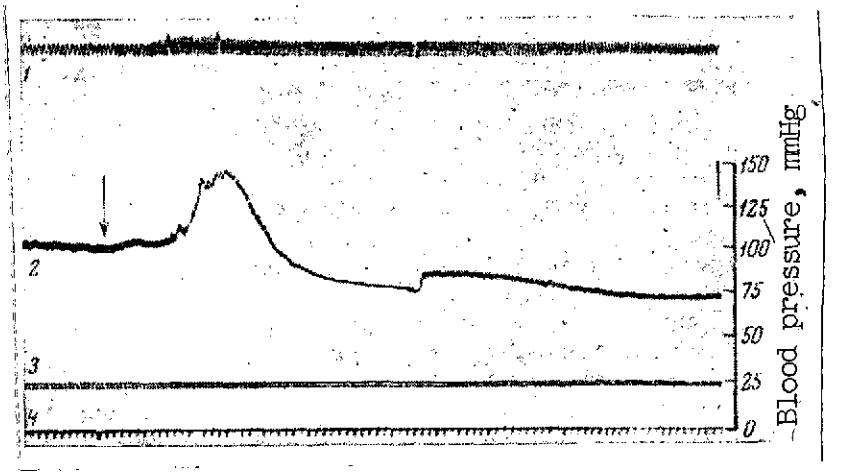

Fig. 65. Changes in respiration, blood pressure and pulmonary ventilation incident to intravenous injection of Korkoniy in a dog weighing 20.5 kg. 1 -- respiration; 2 -- blood pressure; 3 -- volume of pulmonary ventilation (notch, 50 mℓ); 4 -- time, 1 sec. The arrow indicates the intravenous injection of Korkoniy in a dose of 1 mg/kg. Before the Korkoniy injection, the pulmonary ventilation was 8.5 ℓ/min, the volumetric rate of blood flow was 150 mℓ every 1 min 16 sec; after the injection, they were respectively 18.5 ℓ/min and 150 mℓ every 2 min 42 sec.

maintaining its normal pressure and content in the blood and tissues. It is possible that reciprocal interrelations of this kind between respiration and blood circulation are the general rule for all cases of artificial selective stimulation of one of these functions by means of respiratory or cardiovascular analeptics. This question is of independent general physiological interest. But as regards the use of pharmacological respiratory and circulatory stimulants for the purpose of accelerating the organism's desaturation from inert gases, the data we obtained compel us to doubt that such effects are very effective.

Effect of muscular work on the genesis of decompression disorders /219

In contrast to artificial stimulation of the respiration and blood circulation, which always threatens the organism with hypocapnia and, as has just been shown, leads to opposite shifts of these functions, muscular work causes them to increase simultaneously and in harmony with each other and the intensity of

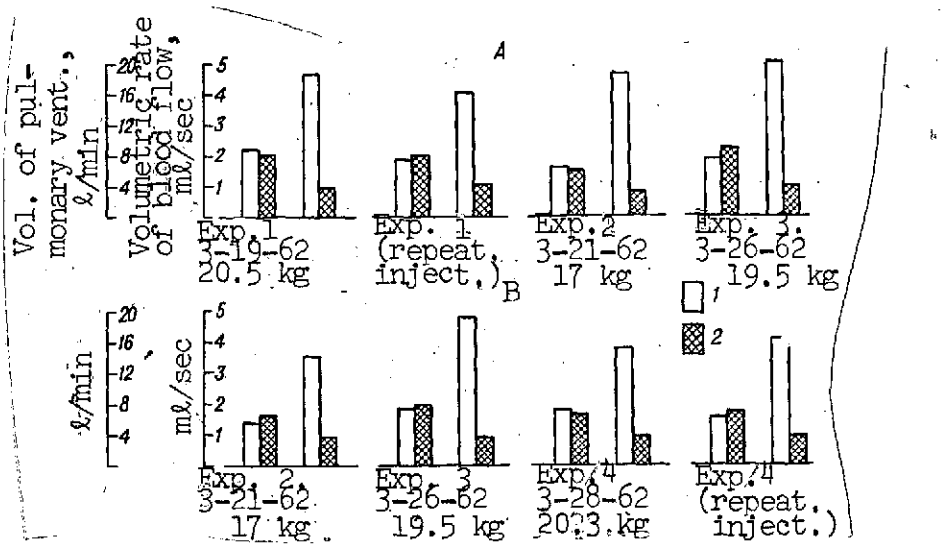

Fig. 66. Changes in pulmonary ventilation and general intensity of blood circulation in dogs under the effect of Korkoniy (A) and Antifein (B). 1 -- volume of pulmonary ventilation; 2 -- volumetric rate of blood flow in whole carotid artery. In each experiment, the left-hand pair of columns are the initial values, the right-hand pair are the values after the Korkoniy or Antifein injection.

exchange processes to increase adequately. Carbon dioxide here emerges as the chief natural stimulant of the respiration and blood circulation. It is natural to suppose that muscular work during decompression from under increased pressure and afterwards must sharply accelerate the organism's desaturation from inert gases and prevent the development of decompression disorders. Actually, however, this question is more complicated.

In diving practice, the idea has taken root that after decompression, complete rest must be observed. This recommendation, which finds its reflection in the rules of the diving service, has some foundation. Cases are known where brisk movements and a physical load have induced decompression phenomena in divers after their return to the surface. In experiments on animals, Whittaker et al [1945] and Harvey [1951] have also shown that muscular contractions at an altitude promote gas formation in the vessels, which, in these authors' opinion, is connected with local drops in pressure in the contracting tissues, leading to formation of cavities and gas seeds, as well as with an increase in the CO_2 concentration.

At the same time, there are indications by a number of authors [Hill 1912; Holden and Priestly, 1937; Yakobson, 1950;

etc] that complete rest during and after decompression is indubitably harmful. In practice, incident to decompression divers generally engage in a light limbering-up at almost every halt. For this there even exists a special command: "Check your joints," which the physician or diving specialist conducting the decompression gives to persons in the chamber during their stay at every halt. Thus, the question of the utility or harmfulness of muscular work incident to decompression from under increased pressure and thereafter remains, on the whole, unclear and requires further study. In this connection, we undertook on two dogs the experiments presented below.

First, in a great number of experiments, we established for each dog that threshold value of increased pressure at which, after complete saturation of the organism with nitrogen and rapid decompression, no decompression disorders developed. For one dog (Zor'ka), this value was equal to 2.6; for another (Valet), 2.4 psig. In experiments involving muscular work, we used a pressure 0.2 atm higher than the indicated threshold value or equal to it. The animals were placed in the recompression /220
chamber and kept there for four hours. After rapid decompression (approximately for 70-80 sec) and the customary walk, the dogs performed certain muscular work running on a treadmill at a speed of 8-10 km/h) for 25 min, after which they were kept under observation for another 2-2.5 hours. In the event that decompression disorders developed, the animal was subjected to therapeutic recompression according to a method that had been tested and had fully proved itself. In control experiments, we used the same pressures with no subsequent physical load. Before the start of the experiments and in the intervals between them, the animals were conditioned to run on the treadmill for 25-30 min daily. While they were running during conditioning as well as in the experiments, we measured their rectal temperature with the aid of thermocouples and a mirror galvanometer. As can be seen from the data of this figure, running on the treadmill after subliminal pressure values did not promote the development of decompression disorders in Zor'ka or aggravate its condition as compared with the control. The use of the same physical load after a stay under a pressure of 2.8 psig (the given pressure value was the threshold for this dog and, in most of the experiments carried out earlier, caused decompression disorders) in one out of two cases was not accompanied by decompression disturbances; nor was any deterioration of the picture observed here as compared with the control.

In all experiments on the dog Valet, we used a pressure of 2.6 psig, 0.2 psig higher than the threshold that always caused decompression disorders. After running on the treadmill in two out of four cases, decompression disturbances did not set in, while all five control experiments terminated in the development of pronounced symptoms and therapeutic recompression.

Thus, the data we obtained show that running on a treadmill apparently does not aggravate, and may even somewhat improve, the prognosis in respect of development of caisson symptoms of the bends type in the dog after emerging from under increased pressure. This assumption must undoubtedly be regarded as very tentative in view of the small number of experiments.

It should be noted that running on the treadmill after emerging from under increased pressure did not come very easily for the animals.

The animal's outward appearance, the character of the panting, the rectal temperature -- all differ sharply from what can be observed in the case of conditioned runs. After decompression, the dog runs with its head held low and its tongue hanging down and gone blue, the typical heat panting is absent and the respiration is heavy and hoarse, with a belabored output at a rate of 40-45 per minute,

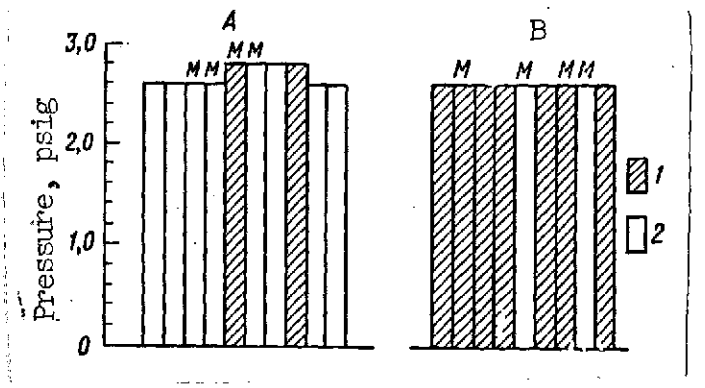

Fig. 67. Results of experiments involving a muscular load (M) for Zor'ka (A) and Valet (B) in the post-decompression period. 1 -- with decompression disturbances; 2 -- without disturbances.

Hyperthermia incident to running after emerging from the chamber attains an extremely high value and appreciably exceeds the customary and constant rise in rectal temperature incident to conditioned running, and its drop at the end of the work occurs more slowly (cf. Fig. 74). All of these manifestations, which are undoubtedly connected with aeroembolism, took place in those experiments involving a muscular load which terminated in development of pronounced decompression disturbances (bends systems), as well as in those where these symptoms were absent. /221

With the pressures we used in our experiments, the organism of the dogs after decompression was in all cases supersaturated with nitrogen above that limit at which gas bubbles begin to form in the venous blood. Inasmuch as this limit was exceeded, running on the treadmill could no longer prevent gas formation, and aeroembolism developed in the dogs with a more or less pronounced intensity.

Under such conditions, those negative factors which have been pointed out by Harvey and Whittaker (change in mechanical relations in the tissues, which promotes the development of gas bubbles, and an intensified CO_2 production which accelerates their growth) could have acted during running. Moreover, in speaking of the effect of muscular work on the development of decompression disorders under the given circumstances, we must not lose sight of the role of deposited blood and lymph in the genesis of air emboli, which has been pointed out by A. P. Brestkin [1952]. If the emptying of the blood depositories that takes place at the start of running, when gas bubbles have not yet had time to form, restricted the development of aeroembolism, then the increased inflow of lymph containing gas seeds into the venous blood throughout the duration of the entire work could have considerably intensified the aeroembolic process. In favor of these hypotheses argues the fact that in experiments with a muscular load, /222
caisson symptoms still develop, as a rule, during the running; but if they passed safely, then they did not arise subsequently. In the control experiments without running, on the other hand, these symptoms appeared at later times.

Moreover, the dogs' running itself was hampered by aeroembolism. Their organism was obliged not only to make provision for harder physical work, but also to contend with such extraordinary and serious factors as gas bubbles in the blood, aeroembolism of the pulmonary capillaries and hypoxemic phenomena. Yet, in spite of such complex conditions, under which one might have expected injury from muscular work, the experiments with running showed no aggravation as compared with the control experiment. Apparently, the powerful stimulating effect that running has on the blood circulation and respiration and, consequently, on the organism's desaturation from the inert gas and on the liquidation of air emboli, transcended the harmful effect of muscular work.

Hence, it follows that muscular work incident to decompression, if it is performed by the organism under the condition of subliminal supersaturation that does not pose the threat of gas formation, can to a great extent accelerate the organism's desaturation from the inert gas.

Effect of an alternating supply of gaseous mixtures on the development of decompression disorders

The effect of alternate inspiration of air and a helium-oxygen mixture (80% He, 20% O_2) during a stay at a depth was studied in crucial experiments on seven dogs. After narcotization (morphine-urethane) and vivisectional preparation, the animals were placed in the chamber and kept at a depth of 90 m for 30 min. With the aid of a hydraulic system (which is described in Chapter 4), their breathing was alternately switched over from air to a helium-oxygen mixture and vice versa. The switching over took 1 min (one experiment), 3 min (two experiments) and 5 min (four experiments). Decompression to the surface was effected either uninterruptedly for 4-5 min (four experiments) or with one 10-min halt at a depth of 20 m (three experiments). Besides these experiments, one experiment was set up with alternate inspiration of air and oxygen at a depth of 90 m. As control we used experiments in which the animals breathed air the whole time. In the experiments, the respiration, pulmonary ventilation and arterial pressure were continuously recorded on a kymograph; in the postdecompression period, a constant watch was kept for gas formation in the vessels of the subcutaneous cellular tissue, thorax, stomach and inguinal region, as well as in the femoral and jugular veins.

The experiments showed that incident to alternate inspiration of air and a helium-oxygen mixture at a depth, the formation of gas bubbles in the blood after decompression is practically just /223 as intensive and violent as it is incident to breathing air. Some experiments created the impression that when gaseous mixtures are alternated, gas formation in the blood at first proceeds even more intensively but, on the other hand, abates somewhat earlier. But, in view of the small number of experiments, there is no point in dwelling on these distinctions. Not to be doubted is the fact that alternation of air and a helium-oxygen mixture yielded no clear positive effect. In such cases with uninterrupted decompression, just as in the control experiments, the dogs generally died from caisson phenomena; and incident to two-stepped decompression, though they survived, they displayed serious respiratory and circulatory disorders. In the experiments with alternate inspiration of air and pure oxygen, the caisson phenomena in the dogs were appreciably weaker, but while the dogs were still at the depth, they developed serious oxygen poisoning, and after decompression, spasmodic phenomena persisted for 3 1/2 hours, right up to the animal's destruction.

Noteworthy is the process of development of oxygen poisoning under these conditions. Not seldom it turns out that incident to the sudden action of high partial pressures of the oxygen, its toxic effect appears immediately after the very first inspirations of compressed O_2; it is said that under such conditions, the oxygen acts with an instantaneous impact, and it is even

supposed that this may, to a certain extent, be based on reflexes from the respiratory tract. The experiment to which we have alluded showed that such ideas do not correspond to the facts.

When the breathing of a dog that had been under a pressure of 10 atm was switched over from air to pure oxygen, no important rapid changes took place in respiration, cardiovascular activity or the animal's general condition. Nor did these changes arise during the entire 5-minute period of breathing pure oxygen, or during the subsequent 5 minutes, when the dog was breathing air again. Only after this at most 10-minute latent period did the air-breathing dog suddenly develop severe oxygen spasms which, in spite of the fact that the animal after this again breathed oxygen at the depth only once in three minutes, lasted several hours after decompression. So the experiment showed that even under very high pressure, oxygen exerts no instantaneous reflex effect; its toxic effect appears only on expiration of a rather long time after the start of O_2 breathing.

Returning to the main results of the experiments, it should be concluded that they confirm the inference drawn by Lazarev and Kurbatov that the organism's supersaturation with inert gases cannot be substantially decreased when these gases are used alternately during the stay at a depth. Clearly, this idea ought to be discarded as being practically ineffectual. But from /224 the standpoint of elucidating the intimate diffusion processes of the different inert gases that are simultaneously present in the organism and the principles of saturation-desaturation of individual tissue zones, those conditions which we are talking about, and the corresponding experiments, undoubtedly conceal much that is of interest. Worthy of mention in this connection is the very recent study of Graves et al [1973], in which the authors discuss the skin affections and serious vestibular disorders that arise in people under a stationary increased pressure when the inert gases in inspired gaseous mixtures are interchanged. They explain these phenomena by the possibility that under these conditions, local foci of supersaturation may arise in the organism because of the counterdiffusion of two different inert gases, and advance a very curious hypothesis to substantiate this possibility theoretically. This hypothesis is conspicuous for its novelty inasmuch as it is a question of supersaturation and the possibility of gas bubble formation in the organism under a stationary pressure merely on account of diffusion relations among inert gases of different compositions when their partial pressures in the inspired air are interchanged. Thus, interchangeable gas supply under a stationary pressure merits attention and further experimental and theoretical study.

Experiments directed at checking another version of alternate supply of different gaseous mixtures that is timed to coincide with different stages of diving were conducted on mice; this

study was carried out jointly with V. A. Aver'yanov and K. S. Yurova.

In a small pressure chamber, the animals were subjected to a pressure of 17 psig for 15 min; then rapid decompression was effected down to 5 psig, at which pressure the mice were kept for 10 min, after which single-step decompression to the surface was effected. These conditions were selected in preliminary experiments in such a manner that, when air was breathed throughout the entire experiment, serious decompression disorders would arise in approximately half of all the cases. The experiments were conducted in four versions: 1) breathing of air throughout the entire experiment; 2) breathing of a helium-oxygen mixture (80% He, 20% O_2) throughout the entire experiment; 3) breathing of air at the bottom and a helium-oxygen mixture at the halt and 4) breathing of a helium-oxygen mixture at the bottom and air at the halt. The air was replaced by the helium-oxygen mixture and vice versa immediately after reaching the intermediate halt by intensively blowing through the chamber while strictly maintaining the pressure at 5 psig. The criterion for the development of pronounced decompression disturbances was pareses, paralyses, spasms, impaired coordination of movements and inability of the animals to maintain their normal body posture. /225

In each experiment, ten mice were placed in the chamber; 100 animals were used in each experiment of each version. The results of the experiments are shown in Table 12, where the figures designating the absolute number of animals correspond simultaneously to percentages.

As can be seen from the adduced data, the results turned out to be different for different versions of the experiment. When using air throughout the entire experiment, pronounced decompression disturbances developed in 55% of the animals and in 41% of the cases, they terminated in death. When using a helium-oxygen mixture throughout the entire experiment, decompression disturbances developed somewhat more rarely (in 44% of the cases), while the death rate was almost half as much (21%) than when using air.

When the animals breathed air at the bottom and a helium-oxygen mixture at the halt, the frequency of development of decompression disturbances was greatest (68%), but the number of fatal cases did not exceed so sharply that which was recorded in the experiments with air. Finally, the last version, in which a helium-oxygen mixture was used at the bottom and air at the halt, stands out sharply from all the rest: decompression disturbances arose only in nine mice and only in two cases did they terminate in the animal's death.

These data can be explained by the different diffusibilities of helium and nitrogen, which determines the different rates of

TABLE 12. RESULTS OF EXPERIMENTS WITH ALTERNATE SUPPLY OF RESPIRATORY GASEOUS MIXTURES, TIMED TO COINCIDE WITH DIFFERENT STAGES OF THE ACTION OF INCREASED PRESSURE AND DECOMPRESSION (100 MICE IN EACH SERIES)

Experiments	Without disturbances	With pronounced decompression disturbances	Death
Air throughout the entire experiment	45	55	41
$He-O_2$ Mixture throughout the entire experiment	56	55	21
Air at the bottom:			
$He-O_2$ mixture at the halt	32	68	42
$He-O_2$ mixture at the bottom; air at the halt	91	9	2

saturation of the organism with each of these gases and of desaturation from them.

This explanation is shown graphically in Fig. 68, which depicts (very hypothetically) the saturation-desaturation curves /226 for each version of the experiments. The curves on the left-hand side of the figure correspond to experiments in which the animals were all of the time either in an air or a helium-oxygen atmosphere. At the end of their stay at the bottom (a pressure of 17 psig), the organism's saturation proves to be greater when using helium than when using nitrogen owing to the fact that helium possesses a higher diffusion rate as compared with nitrogen. By virtue of the same circumstance, when the animals are transferred to the intermediate halt (a pressure of 5 psig), the helium leaves the organism faster than the nitrogen; as a result, at the time of final decompression to the surface, the organism's saturation with helium proves to be less than its saturation with nitrogen. In conformity with this, after rapid transference of the animals to normal pressure, the degree of the organism's supersaturation with nitrogen (cf. columns in the lower part of the figure) proves to be greater than its degree of supersaturation with helium.

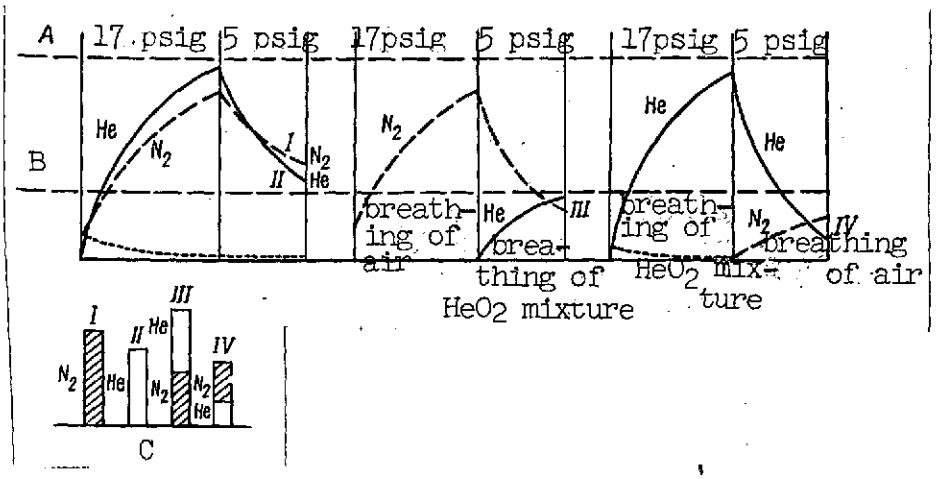

Fig. 68. Saturation of the organism of mice with nitrogen and helium and its desaturation from these gases in different versions of the experiments (hypothetically). A -- complete saturation at 18 atm; B -- complete saturation at 6 atm; C -- degree of supersaturation after termination of decompression.

The curves depicted in the middle part of the figure correspond to experiments in which the animals breathed oxygen at the bottom and a helium-oxygen mixture at the halt. Here, at the halt, the nitrogen pressure in the organism drops for an understandable reason faster than in the control experiments with uninterrupted breathing of air. But the place of the departing nitrogen is taken by the helium, and owing to its high diffusion rate, its pressure by the time of decompression to the surface will have succeeded in growing to significant values. In this version of the experiments, the total supersaturation of the organism with helium and nitrogen upon the animals' return to normal pressure proved to be greatest. In the right-hand part of the figure are depicted the curves for the optimum version of the experiments, in which the helium-oxygen mixture that the /227 animals breathed at the bottom is replaced by air at the halt. In this case, the readily diffusing helium escaped rapidly from the organism, and its pressure at the end of the experiment dropped very sharply; but the pressure of the nitrogen, owing to its slow entry into the organism, increased not so significantly. The organism's total supersaturation with the two gases under these experimental conditions turned out to be least.

The frequency of development of pronounced decompression disturbances in the animals in the different versions of the experiments conforms directly to the supersaturation values

depicted in the figure. But as far as the death rate is concerned, another interesting circumstance comes to light. In those cases where supersaturation takes place with helium alone (version II) or where it is determined chiefly by helium (version III), the animals die of decompression disorders considerably more rarely than incident to supersaturation with nitrogen (version I). Thus, in the case of supersaturation with nitrogen, 41 of the 55 animal casualties died (74.5%); in the case of supersaturation with helium alone, 21 out of 44 died (47.7%); and in the case of supersaturation chiefly by helium, 42 out of 68 died (62.4%). These facts agree nicely with the data of A. P. Brestkin mentioned earlier and with the results of our experiments with intravenous injection of helium and nitrogen. Helium, as has been established by Brestkin, forms bubbles at lower degrees of supersaturation than nitrogen; but, as our experiments have shown, helium emboli exert on the organism a less noxious effect than nitrogen emboli. Now then, how can the above-discussed methods of increasing the effectiveness of decompression disorder prophylaxis be evaluated?

The experiments we conducted compel us to suppose that the use of drugs is hardly a promising way of improving decompression disturbance prophylaxis. Thus, the experiments with Korkoniy and Antifein showed that artificially induced panting not only does not reduce the danger of decompression disturbances developing, but even somewhat increases the frequency of their development. This can be explained by the fact that incident to the action of Korkoniy and Antifein, the general intensity of the blood flow decreases in proportion to the increase in pulmonary ventilation.

There is every reason to believe that powerful artificial stimulation of the blood circulation will not yield a positive effect incident to decompression effects, either: it is probable that it must always be accompanied by a greater or lesser inhibition of respiration directed at retaining carbon dioxide in the organism. Actually, if we take the example of so powerful a stimulant of the blood circulation as adrenaline, its pronounced effect on the cardiovascular system is always accompanied by a clear inhibition of respiration. It is possible that this is connected with the patently negative effect of adrenaline on /228
the course of artificial and decompression aeroembolism that we established in a separate series of experiments on rabbits.

Pharmacological stimulation of the blood circulation incident to decompression effects is fraught with still another danger. Incident to such stimulation, together with an increase in the general intensity of the blood circulation and blood supply of most of the organs, there generally takes place a restriction of the blood flow in individual parts of the organism. In these parts, therefore, especially favorable conditions for gas formation are created, which may not only cause local functional

disorders, but may also be a spur to intensification of general aeroembolic phenomena.

Simultaneous powerful pharmacological stimulation of the respiration and blood circulation, provided it can be achieved, will clearly never be of long duration inasmuch as it must inevitably lead to acute hypocapnia. But hypocapnia, apart from the fact that it can by itself bring the organism out of the state of equilibrium, in the case of decompression effects is doubly undesirable: ultimately transcending pharmacological effects, it can cause an inhibition of respiration, stenosis of the vessels and reduction in the intensity of blood flow, i.e., functional shifts that are directly opposed to those which are characteristic of the organism's natural defense reactions incident to the development of decompression phenomena and for which the use of pharmacological preparations was intended.

The heuristic experiments using caffeine and lobeline that were presented above give grounds to suppose that simultaneous artificial stimulation of the respiration and blood circulation, provided it is not excessive, may be of some use in cases of single-step decompression. It is to such cases, we must imagine, that the prospect of practical utilization of pharmacological stimulants of cardiovascular activity and respiration for decompression disorder prophylaxis will be confined.

In connection with the data obtained, the thought arises that for artificial intensification of the defense reactions of the respiration and blood circulation incident to decompression effects, it might be more expedient to test not immediate stimulants of the above-mentioned functions, but substances which intensify exchange processes and stimulate, in virtue of this, the activity of the cardiovascular system and the respiratory apparatus. In general, however, incident to decompression effects, as well as other unusual external effects, the problem of pharmacological stimulation of defense-adaptive reactions, which at first glance seems very tempting and promising, turns out to be, when put to the test, not so encouraging. Artificial intensification of adaptive reactions generally has its negative side. The surest and most reliable means of intensifying and improving defense-adaptive reactions is the rationally planned conditioning /229 of the organism to the action of extraordinary factors. Clearly, decompression effects are subsumed under this rule. The question of the effect of muscular work on the organism incident to decompression effects has, in our opinion, an incomparibly greater practical significance than that of the utilization of pharmacologic substances.

Undoubtedly useful for desaturating the organism from inert gases and ridding it from air emboli is simultaneous and proportionate intensification of respiration and blood circulation, which is natural for the condition of muscular work. This

usefulness, under the conditions of our experiments, in a number of cases exceeded the harm that muscular activity might entail by accelerating the process of gas-bubble formation. It may be that this latter, negative role of muscular activity, which comes clearly to the fore incident to decompression effects in a rarefied atmosphere, is of less significance in the increased pressure range. It should be noted that the question of the usefulness or harm of muscular work incident to gradual decompression from under increased pressures goes hand in glove with the principal problem of decompression disorders -- the conditions and principles of formation of gas bubbles in the organism, incident to its supersaturation with an inert gas. If we assume that bubbles arise only for a certain limit of supersaturation, then we can suppose that muscular work incident to decompression permitting only subliminal supersaturation of the organism, by accelerating its desaturation from the inert gas, makes it possible to reduce the duration of decompression without bringing in its wake the danger of decompression disorders developing.

As regards the experiments with alternation of inert gases under stationary increased pressure, they do not, as has already been mentioned, hold out any practical promise for decompression disturbance prophylaxis. But the experiments involving the use of nitrogen and helium at different periods of the experiment give good grounds to assert that rational alternation of inert gases that is timed to coincide with different stages of diving may be a very effective means of decompression disturbance prophylaxis. This conclusion, since the publication by H. Keller and A. A. Bühlman [1965] has lost its novelty. It turned out that Keller's sensational feats of diving to great depths with relatively short decompression times was based precisely on the principle of alternate supply of respiratory mixtures containing different inert gases timed to coincide with different stages of diving. It can be said that Keller in his unremitting struggle to conquer great depths opened up new paths and prospects in this region. The principles of deep sea diving that he developed jointly with Bühlman are now widely implemented. They are /230
bearing fruit in new achievements, in witness whereof are the successes of the French firm Comexx, which has been organizing dives to depths of 400-500 m and deeper.

On the effectiveness of different therapeutic decompression methods

As has already been pointed out, in all cases where bends symptoms develop in dogs, the animals were subjected to therapeutic recompression. In the course of the entire study, more than 170 cases of development of bends symptoms in dogs were traced, and therapeutic recompression was consequently effected the same number of times. At first it was effected according to

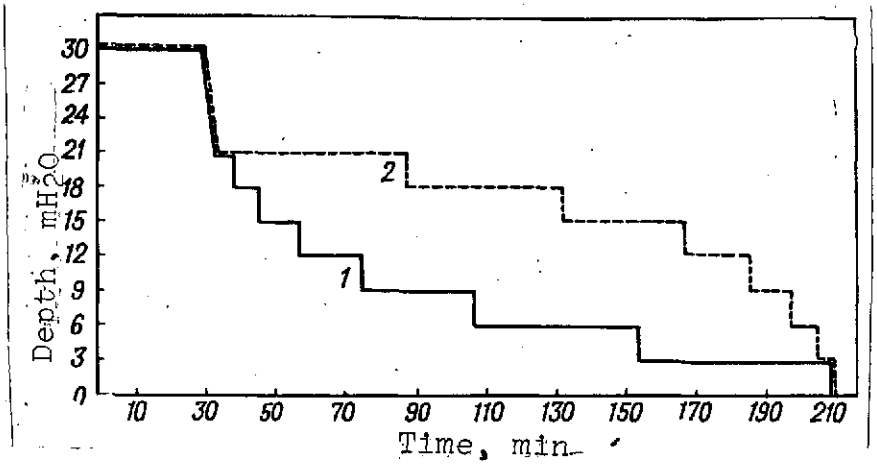

Fig. 69. Barograms of therapeutic methods. 1 -- method calculated according to diving service rules; 2 -- empirical method employed in most of our experiments.

a method calculated by V. A. Aver'yanov on the basis of a procedure that is current in diving practice but with allowance for the fact that the dog's organism is desaturated from nitrogen more rapidly than the human organism. Inasmuch as the depths used in the initial experiments were shallow -- on the order of 20-30 m -- and the question was one only of bends symptoms that are classified in diving practice as being comparatively light, the maximum pressure adopted for therapeutic recompression was equal to 3 psig, while the holding time under this pressure was equal to 30 min. The first halt was effected at a depth of 21 m (a pressure of 2.1 psig); it was the shortest. Subsequent halts were effected every 3 m, and the staying time at each one of them kept being lengthened. A barogram of the method is shown in Fig. 69 by the solid line. In experiments involving determination of the coefficient of safe supersaturation for animals, this method, at pressures of 2.0-3.0 psig, generally/231 ensured a good enough therapeutic effect. Cases of incomplete liquidation of the bends symptoms were extremely rare exceptions. Subsequently, however, when we began using a pressure of 4.5 psig in the experiments, such cases became more frequent. By this time, it had become clear from experiments in which decompression phenomena were induced by means of rarefaction that the gas bubbles which had been formed in the organism and which were causing the bends symptoms were characterized by a high stability and were liquidated extremely slowly. All of this gave grounds to use a different therapeutic method. This method was based

on no calculations, but only on the consideration that for desaturation of the bubbles that cause bends symptoms, it was more efficient to increase the staying time under high pressure, while exposure to lower pressures could be reduced inasmuch as the supersaturation corresponding to them was known to be less than the maximum permissible one. Proceeding from what has been said, we used the method whose barogram is shown in Fig. 69 by the dotted line. This method, as can be seen from the barogram, is the mirror image of the method calculated by V. A. Aver'yanov: here, in contrast to the calculated method (which was similar in character to those utilized in practice), the staying time at the first halt was the longest, while those at subsequent halts were steadily reduced.

To compare the efficiency of the above-mentioned two methods, we undertook a special series of experiments in which dogs were kept under a pressure of 4.5 psig for 35-45 min and then subjected to rapid decompression to normal pressure. For each animal (five dogs were used in the experiments), we used deliberately dangerous staying times under pressure, times which ensured the development of bends symptoms after decompression. Immediately after the appearance of the symptoms, the animals were subjected to therapeutic recompression either by the calculated method or by the empirical method of opposite character. In those cases where, as a result of therapeutic recompression, the bends symptoms disappeared entirely, the dogs were raised to an altitude of 4.5 km one hour after emergence from under pressure, and if the symptoms did not appear here in the course of 5 min, to an altitude of 6 km. This made it possible to decide whether latent gas bubbles remained in the organism after outwardly effective therapeutic recompression. The experiments yielded the following results:

	Number of experiments	Complete liquidation of symptoms	Incomplete disappearance of symptoms	Number of subsequent ascents to an altitude	Reappearance of symptoms
Calculated method	23	19	4	19	10
Empirical method	24	23	1	23	5

The adduced data show, firstly, that the immediate effect /232 of therapeutic recompression according to the calculated method is less than that from recompression according to the empirical method. With the calculated method, out of 23 cases of therapeutic recompression, bends symptoms were kept in four; with

the empirical method, out of 24 cases, bends symptoms were kept only in one. Secondly, and this is more important, the data show that after using the calculated method, latent gas bubbles were kept in a considerable number of cases. With the calculated method, inducement of decompression phenomena by means of ascents to an altitude disclosed the presence of latent bubbles in ten cases out of 19, while with the new method of opposite character, it did so in five cases out of 23. Basing ourselves on the results of these experiments, in subsequent investigations we began making use of this new method, adding to it, in those cases where pressures of 4.5 psig were used, initial compression up to 5 psig with a 2-min exposure there; then we went over to a pressure of 3 psig, and kept on adhering to the indicated method. Out of 130 cases of therapeutic recompression conducted in this manner, only in six was incomplete liquidation of the symptoms noted.

Thus, the idea of increasing the exposure to high pressures completely demonstrated its value. It should be noted that lenghtened exposures to high pressures leads to more rapid complete desaturation from gas bubbles, while long exposures to lower pressures cause only a reduction in volume of these bubbles down to a size at which they cease to cause bends symptoms. The adduced data indicate that saturation and desaturation calculations for the organism's different tissues in the process of therapeutic recompression are very approximative and far from accurate. Here we must recall those cases where, incident to therapeutic recompression under the method in use at the present time, the physician enters the chamber with the casualty, passes the entire so-called course of treatment there and, upon its termination, i.e., upon emergence from the chamber, it is discovered that this physician has developed pronounced bends symptoms.

It follows from all that has been said that, even more so than prophylactic methods, the therapeutic methods adopted for use need experimental checking, elaboration and, perhaps, even radical alteration. The results of the experiments discussed in the present chapter lead to the following conclusions.

According to heuristic data, the moderate stimulation of heart action and respiration caused by subcutaneous injection of a caffeine and lobeline mixture can reduce the danger that decompression disorders will arise upon transition from increased pressure to normal pressure.

Intensive artificial stimulation of the respiration by means of Korkoniy or Antifein during the organism's desaturation from nitrogen in no way prevents development of decompression /233
disturbances for the reason that, simultaneously with the increase in pulmonary ventilation and in proportion to this increase,

the general volumetric rate of the blood flow decreases. This reaction of the organism is apparently directed at maintaining the normal pressure and total amount of carbon dioxide in the blood and tissues.

According to heuristic data, muscular work in the postdecompression period (after a stay under increased pressure), even in the presence of aeroembolic phenomena in the organism, does not aggravate the danger that decompression disturbances will develop.

In conformity with the theoretical conclusions of Lazarev and Kurbatov, alternate inspiration, during a stay under increased pressure, of gaseous mixtures containing different inert gases does not prevent development of decompression disorders.

Alternate supply of respiratory gaseous mixtures that is timed to coincide with different stages of diving is a promising means of decompression disturbance prophylaxis.

The therapeutic recompression-decompression methods used in diving practice need revision. Sufficiently effective therapeutic methods must be thoroughly grounded in theory and experiment.

CHAPTER 12

PRINCIPLE RESULTS OF PHYSIOLOGICAL ANALYSIS OF DECOMPRESSION DISORDERS ARISING IN THE ORGANISM AFTER A STAY UNDER INCREASED PRESSURE

Summing up the results of the investigations we carried out, it is necessary first of all to dwell on the results of experiments directed at studying the principal condition under which decompression disorders arise. In conformity with Haldane's old conclusions, these data showed that the organism's maximum permissible supersaturation with an inert gas, which determines the development of decompression disorders, depends not so much on the absolute magnitude of the pressure drop as it does on its rate.

The real measure of maximum permissible supersaturation, as ensues directly from these experiments, is a coefficient, i.e., the ratio of the pressure of the inert gas in the organism to the total surrounding pressure after decompression, and not the /234
difference between the pressure of the gas in the organism's media and the manometric pressure, as proponents of the idea of

the "limit" have supposed. In checking this idea, it became clear that its authors were mistaken not only about this important point of principle, a point of primary practical significance. Another mistake they made was to assert that the "limit" in individuals is constant; in other words, that the organism's resistance to decompression disturbances is determined exclusively by the ability of its tissues to retain in solution the inert gas with which they are supersaturated and that it is not subject to any substantial variations. This assertion reflects the erroneous, but nevertheless rather wide-spread opinion that the appearance of bubbles in the organism directly determines the development of decompression symptoms, in particular the bends symptoms. The pronouncements that one encounters to the effect that "silent" gas bubbles may exist in the organism have not been attracting the due attention of researchers, and many of them continue to adhere to simplified ideas -- if gas bubbles have arisen in the organism, then specific decompression symptoms must of necessity develop, and if there are no such symptoms, there are no bubbles. Although these ideas are not always formulated in so direct and categorical a form, it follows from the arguments and logical constructs of many authors of experimental studies and textbooks, as well as practicing physicians who safeguard diving operations, that these ideas are regarded as being incontestible and self-evident.

Experiments involving direct determination of initial gas formation in the blood of anesthetized animals, as well as experiments in which caisson phenomena are induced by means of ascents to an altitude, have shown in all clarity how far these ideas are from the truth.

Gas formation begins in the venous blood after a stay at depths that are known to be safe in respect of development of pronounced decompression disturbances, in particular the bends symptoms; it always and of necessity precedes the development of these symptoms. In their initial period, aeroembolic phenomena take a masked and latent form; the effect of inconsiderable rarefaction makes them manifest at once. At a certain stage in the latently progressing aeroembolic process, gas bubbles are indeed formed in the organism which are responsible for the development of bends symptoms; but, apparently, these bubbles, in virtue of their insufficient size, very often do not lead to development of these symptoms, either; the latter develop only in those cases where the bubbles attain a certain size or where the overpressure of the gas of which they consist is increased by a certain amount.

Thus, the organism's resistance to decompression disturbances is determined by the coefficient of safe or permissible supersaturation that depends to a considerable extent on purely physiological conditions. This physiological coefficient is always /235

higher than the physical coefficient of maximum permissible supersaturation that determines the appearance of gas bubbles that are visible to the naked eye in the organism's media and, in particular, its blood; it differs sharply in different animals, is not uniform for different people, and is subject in every individual to great fluctuations; it increases regularly with gradually intensifying, repeated, decompression effects and can decrease considerably as a result of these effects, if they are excessively strong, as well as of other effects that change the organism's physiological condition. The coefficient of safe or permissible supersaturation therefore corresponds to that degree of supersaturation of the organism at which there always exist gas bubbles in its internal environment, but at which these bubbles, owing to their small size and number, do not yet lead to development of those specific symptoms which it is customary to regard as the criterion for development of decompression disorders.

It becomes clear from what has been said that practical application of the idea of the "limit" may be fraught with serious unfavorable consequences, which indeed was already brought to light in part when the first attempts were made to use it. First of all, decompression methods calculated according to the principle of the "limit" proved to be unreliable in their very foundation. In essence, they ought to have been incomparibly longer than methods calculated according to the coefficient.

To avoid lengthening the methods, authors have been obliged to resort to a number of speculative conclusions: thus, for example, they consider that tissues that become rapidly desaturated (what they evidently have in mind in the first place is the blood) possess a particularly great ability to retain nitrogen in supersaturated solutions; in these tissues, as has been proposed by A. N. Bukharin, gas bubbles are not formed even incident to fourfold supersaturation, which clearly does not correspond to the experimental facts. But if, without giving up the idea of the "limit," authors do succeed in obtaining, with the aid of such artificial assumptions, decompression methods that are acceptable from the standpoint of duration, the danger for practice remains and resides in the principle which is put forward for using these methods.

As has already been said, authors propose to determine the "limit" in divers and, depending on its value, to divide them into separate groups; proceeding from the idea that the "limit" is constant, they recommend that their more or less drastic methods be used for each such group. The facts, however, show that there cannot even be any question of the constancy of the "limit;" the organism's resistance to decompression disturbances is variable and may increase considerably under some conditions and decrease under others. In divers who have shown little /236

resistance incident to a first determination of the "limit," the resistance may, in the course of the study, increase, and the use in their case of decompression methods that are dragged out in time may prove to be unwarranted and unnecessary. Conversely, in divers with a high "limit," the resistance may drop sharply under the effect of conditions of one sort or another, and when the particularly drastic methods intended for them are used, the development of serious decompression disorders will be inevitable.

Thus, the revision of the established principles of calculating and implementing decompression methods that have been undertaken by I. I. Savichev and A. N. Bukharin is groundless, and the practical suggestions they have put forward are unacceptable. But this does not gainsay the existence of individual differences in people's resistance to decompression disorders.

The investigations we carried out have led to the unshakeable conclusion that aeroembolic phenomena play a leading role in the development of decompression disorders after a stay of the organism under increased pressure. As is clear from analysis of the literature, no author rejects out of hand the possibility that postdecompression aeroembolism may develop and, on the other hand, the majority of researchers even point to its probable very great significance in the development of decompression disturbances. But, in spite of all this, in the available studies the true role of aeroembolism in the development of decompression disorders has not been disclosed or clarified to the proper extent. In the overwhelming majority of investigations, an equal weight is ascribed to the probability that gas bubbles will be formed in the blood and that they will be formed in the tissues. Authors, often with no direct experimental data at their disposal, are inclined to assign the leading role either to intravascular or to tissue gas formation in the development of various decompression symptoms, or consider them equally probable. Thus, in the literature concerning the comparative significance of postdecompression gas formation in the blood and in the tissues, ever more contradictions are accumulating. Researchers who argue in favor of the predominant role of aeroembolic phenomena have their divergences, too.

In concentrating their attention on aeroembolism of the venous system and pulmonary vessels, authors, when they pass to explication of local symptoms or central nervous postdecompression disorders, very often suppose that they are based on arterial aeroembolism. Some researchers, for example, N. I. Yakobson, even connect the entire pathogenesis of decompression disturbances primarily with gas formation in the arterial blood. Thus, those authors who acknowledge the leading role of the aeroembolic process in the development of decompression disorders, share no clear ideas about the localization, or prevalence or

nature of aeroembolic phenomena. Finally, in all investigations /237
concerning postdecompression aeroembolism, the latter is regarded only as the immediate cause of the pathologic disturbances occurring in the organism, and no author so much as raises the question: what is the significance of aeroembolic phenomena in the development of the organism's compensatory defense reactions?

Our investigations permit more light to be thrown on the question of postdecompression aeroembolism.

They show that the development of decompression phenomena in the organism first and foremost reduces to aeroembolism of the venous system and pulmonary capillaries. At the same time, the aeroembolic process, the development of which in all of its details has been analyzed in the course of expounding the factual material, emerges as the obligatory and dominant phenomenon incident to decompression disturbances of any intensity, beginning with their mildest manifestations and ending with their fatal ones. Of physiological significance in this process, so out of the ordinary in character, are chiefly three factors. Of primary significance is the plugging up of the pulmonary capillaries with air emboli, which leads to changes in the gaseous composition of the arterial blood and in the hemodynamics and causes by means of these physiological shifts those responses on the part of the respiration and cardiovascular system which themselves determine to a considerable extent the entire subsequent development and final outcome of decompression disorders. The leading role of this fundamental factor is directly confirmed by the fact that artificial aeroembolism caused by intravenous air injection always completely reproduces the general picture of decompression disorders and causes the very same reactions of the respiration and blood circulation.

Playing the role of second physiologically significant, not to say very important, factor in the process of postdecompression aeroembolism are local aeroembolic phenomena in the capillaries of different regions of the greater circulatory system. They evoke regional vascular responses, cause the development of local symptoms (in particular, itching, pains and, in serious cases, cerebral symptoms) and, in those cases where they develop in extensive regions of the peripheral vascular network, influence also the general conditions of blood circulation. Especially favorable conditions for the development of aeroembolic phenomena in the peripheral ramifications of the greater circulatory system are created incident to significant degrees of supersaturation of the organism with nitrogen when, soon after decompression, not only the venous blood, but also the arterial blood proves to be supersaturated, and fine gas bubbles appear in it, too. In this case, the process of gas formation in the peripheral vascular ramifications are shifted ever more toward the arteries

and create all the prerequisites for complete embolization of the corresponding vascular zones.

Finally, the third factor of significance in the process of /238
pronounced decompression aeroembolism is the very fact of the filling up of the vascular system with gas bubbles. When their number is considerable, this leads to an increase in the total volume of blood and gas filling the vascular system and, when gas formation is still more intensive, to the impossibility of blood circulation owing to purely physical causes.

Incident to decompression aeroembolism, there are always two processes going on simultaneously in the organism: formation of gas bubbles in the venous blood flowing out of the tissues and their liquidation in the lungs.

It is only in virtue of this liquidation of air emboli that is constantly going on that the organism proves able to endure severe caisson phenomena, in the course of whose development an enormous amount of gas is formed in the blood, as well as successive intravenous injections of large doses of air. The reactions of respiration and blood circulation and the operating conditions of these systems play a decisive role in the liquidation of air emboli. So strained and precarious is the equilibrium between the processes of gas-bubble formation and liquidation incident to some decompression effects -- in particular, rapid ascent from great depth -- that the most short-lived inhibition of the respiration and heart action prove to be enough for the process of bubble formation to get the upper hand, and the organism is foredoomed to death.

Connected with the aeroembolic process, as has already been pointed out, are changes in the respiration and blood circulation that are typical of decompression phenomena. Little attention is paid to these functional shifts because they do not constitute anything specific. At the same time, they are the earliest indications that caisson phenomena have developed in the organism and are the constant and obligatory companions of pronounced decompression disorders. The specific decompression symptoms (itching skin, bends symptoms, etc), on the basis of which it is customary to judge of the presence or absence of caisson phenomena in the organism, are less constant, do not always arise and themselves define an already definite stage in the development of these phenomena, whereas the reactions of the respiration and blood circulation betoken the beginning of the process and reflect its entire dynamics. Thus, the absence of specific symptoms does not yet mean that caisson phenomena are absent from the organism; but the presence of these symptoms undoubtedly attests to the fact that even before they appeared, the aeroembolic process had begun in the organism. Hence it follows that, as a rule, all of the current prophylactic

decompression methods that have been worked out on the basis of the generally accepted criteria -- specific decompression symptoms -- are no guarantee against the development of aeroembolic phenomena in divers.

It should be noted that this conclusion, to which we were led by our experiments almost as long as ten years ago, has received some confirmation in a recent study by A. Evans et al [1972]. With the aid of an ultrasonic pick-up placed on the thorax below the region of the heart in examinees, the authors noted signals attesting to the presence of gas bubbles in the blood -- even in the case of stepped decompression -- in the absence of any symptoms. Should the above-mentioned conclusion be finally confirmed, another big question would arise: should existing prophylactic methods be changed in such a manner as to guarantee the absence of gas formation in the blood, or should the earlier principle be adhered to, and, in consideration of the organism's powerful defense reactions, should it be left up to the organism to cope with aeroembolic phenomena on its own? If this question were to be solved in the first manner, there would be reason to check the expediency of using, incident to decompression, muscular work as a natural powerful stimulant of the respiration and blood circulation that sharply accelerate the organism's desaturation from the inert gas. /239

One of the main results of the work we have done is that it has shown the organism's ability to actively resist the development of caisson phenomena and to increase its resistance to decompression disturbances incident to repeated intensification of decompression effects.

Even earlier it was known to practicing physicians safeguarding diving operations, as well as to specialist physiologists, that the danger of decompression disturbances developing in apprentice divers is greater than it is in professional divers, and that the experience of working underwater certainly influences man's susceptibility to decompression disorders. According to the generally accepted opinion, however, this circumstance was connected with the diver's habituation to the whole complex of unusual conditions encountered in diving, with his lesser agitation on successive dives and the lesser (as compared with his first dives) saturation of his organism with inert gases during his work at the bottom. The question of any active adaptation of the organism to caisson phenomena or specific conditioning to the effect of gas bubbles forming in the blood was not so much as raised. The absolute unusualness of the factor in question, the fact that it had, in the final analysis, a purely physicochemical cause -- the maximum permissible supersaturation of the blood and tissues -- and, finally, ideas about the inevitable development of decompression symptoms

incident to the appearance of bubbles in the organism -- all of this seemed incompatible with the possibility of the organism's active adaptation and specific conditioning to caisson phenomena.

The facts showed that this was not so. The organism's powerful defense resources in respect of decompression disturbances appear clearly as early as its first encounter with the factor causing them -- gas bubbles appearing in the blood --, /240 even in those cases where this factor is distinguished by its high intensity, i.e., when gas formation is very intensive. And incident to repeated decompression effects of threshold intensity, these defense resources become stronger.

The organism's defense against the harmful effect of decompression aeroembolism is organized according to an original principle. Inasmuch as gas bubbles in the blood are an absolutely unusual factor that has never been encountered in the course of evolution, it is natural tnat the organism should possess no physiological mechanisms that might be set in motion by the direct and immediate agency of this factor. But as soon as this factor attains those values (size and number of bubbles being formed in the blood) at which physiological shifts arise that can be perceived by the organism's neural apparatus, answering reflex reactions are immediately evoked. The principle physiolgical shift that occurs incident to the development of caisson phenomena, and the one that is of greatest significance to the organism, is changes in the gaseous composition of the arterial blood: an increase in carbon dioxide pressure and content and a decrease in oxygen pressure and content.

Both of these physiological indices are extremely delicate, and accurately and thoroughly regulated by the organism by means of reflex reactions on the part of the respiration and cardiovascular system that were worked out in the course of evolution first of all in connection with muscular activity, which was and is one of the chief active forces of the evolutionary process.

Decompression aeroembolism, which causes arterial hypercapnia and hypoxemia because of the plugging up of the pulmonary capillaries with gas bubbles, is what elicits these reflex reactions; they play a decisive role in the process of liquidation of air emboli; they intensify the saturation of the tissues from the inert gas and thereby prevent or, at any rate, limit the formation of new gas bubbles in the blood.

Incident to strong decompression effects, when aeroembolic phenomena are attaining a considerable intensity, to the changes in the gaseous composition of the blood are added general hemodynamic shifts caused by the belabored passage of the blood through the pulmonary vessels, as well as through the vascular ramifications of the greater circulatory system, in those

regions where the process of gas formation is proceeding particularly energetically in the peripheral blood. These general hemodynamic shifts are an adequate stimulus for those reflexes of self-regulation of the blood circulation which sustain its sufficient general intensity. Thus, with an intensification of the active factor, i.e., intensification of aeroembolic phenomena, the organism's defense against their noxious effect is also intensified. These two general consequences of post-decompression aeroembolism -- changes in the gaseous composition/241 of the arterial blood and shifts in the hemodynamics -- also emerge as the triggering mechanism of adaptive defense reactions incident to the development of caisson phenomena in the organism. Thus, the organism's defense in the case in question is organized not in terms of direct responses to the unusual factor acting on it, but in terms of those reactions which are caused by the most immediate physiologically significant consequences of this factor.

It must be supposed that this principle underlies all cases of interaction between the organism and new factors that have not been encountered in the course of evolution, and it is clear that owing to this principle, the organism is not left defenseless in the face of even the most extraordinary external effects and displays the ability to adapt to them to one or another extent. In this connection, we must recall P. M. Al'bitskiy's pronouncement reflecting the general views of the founders of domestic physiology and pathology about the organism's ability to resist different external effects. Analyzing the phenomenon of reverse action of carbon dioxide which he discovered, P. M. Al'bitskiy wrote: "But whatever the harmful effect that has acted on the organism, provided it has not caused immediate death and the life of the injured organism still continues if only for some time, together with the above-mentioned (pathological phenomena -- P. G.), there always and without fail develop phenomena of a quite different order and different biological significance. These are phenomena directed, on the one hand, to limiting the further effect of the destructive influence and, on the other, to weakening and eliminating those chemical, structural and functional disorders which have already been brought about by this influence."[16] And further: " The ability of self-defense is the most important condition for the life and resistance of organisms; it is a radical property of protoplasm, a fundamental biological law."

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[16] P. M. Al'bitskiy. "On the reverse action or 'consequence' of carbon dioxide and on the biological significance of the CO_2 usually contained in the organism." Information and Biographic Bureau, 1911, pp. 64-645.

[17] Ibid.

The organism's ability to actively resist the development of caisson phenomena and increase its resistance to decompression disorders is still another confirmation of those important conclusions of principle which make it possible to count on the organism's adaptation to the most diverse extraordinary effects, perhaps even such as are not in question presently, but which mankind will encounter in the future.

The results of our physiological analysis of the conditions under which decompression disturbances develop and the mechanism which governs them have, in our opinion, a direct connection with the practice of diving and caisson work.

In the first place, they compel us to pay close attention,/242 incident to a man's emergence from under increased pressure, to such signs as barely noticeable shifts in the respiration, changes in the pulse rate, changes of the organism's general condition and sensations of breakdown and fatigue inasmuch as all these signs are signals of decompression phenomena that are beginning in the organism.

In the second place, they oblige us to deal very carefully with all of the specific mild (and, as it has been customary to consider them, local) decompression symptoms inasmuch as the development of these symptoms always takes place against the background of a general aeroembolic process.

In the third place, they compel us to deal quite specially with all cases of rapid single-step decompression after short exposures to high pressures. In these cases, owing to the fact that in the early period after decompression it is not only the venous, but also the arterial, blood that proves to be in a state of supersaturation, the threat of the sudden development of serious decompression disorders is always present.

Finally, in the fourth place, -- and this is practically the most important -- the results of our work disclose additional possibilities for decompression disorder prophylaxis. They show that in addition to the working out of those optimum external conditions during work under pressure during the decompression period and thereafter under which the threat of development of decompression disorders would be reduced to a minimum, there is still another way of increasing the safety of diving and caisson work that is connected with the possibility of increasing the organism's resistance to decompression disturbances. Data we have obtained point to the necessity of working out such a scientifically well-founded regulation of diving and caisson work as would ensure the conditioning and increased resistance of the organism to decompression disorders, which are the chief danger for man engaged in these forms of activity.

The principal results of the above-described experimental studies can be summaried as follows.

Experimental checking of the idea of the "limit" showed that this idea is untenable and that the practical notions ensuing from it are unacceptable. The true measure of the organism's supersaturation with an inert gas is the coefficient of safe supersaturation, whose value depends on physiological factors and is subject to considerable fluctuations. The physiological coefficient of safe supersaturation of the organism always considerably exceeds the physical coefficient of supersaturation of the blood, which determines the start of the formation of gas bubbles in it.

The main and decisive link in the mechanism of development of decompression disturbances is aeroembolism of the venous system and pulmonary capillaries. It always precedes the appearance of bends symptoms and accompanies the development /243 of pronounced decompression disturbances; the changes in respiration and cardiovascular activity that it causes are earlier and more constant signs of decompression disturbances than the specific decompression symptoms.

In addition to the appearance of gas bubbles in the moving venous blood, very often formed incident to decompression effects that do not cause manifest decompression disturbances are latent gas bubbles -- potential causative agents of bends symptoms -- that are localized at certain points. They are distinguished by high stability and can exist in the organism for a long time without causing any functional disorders.

The organism possesses powerful defense resources in respect of decompression disorders, which permit it to actively resist their development and to adapt to the effect of such an absolutely extraordinary factor as gas bubbles appearing the blood. Incident to repeated intensification of decompression effects, the organism's resistance to decompression disturbances increases considerably.

The increase in the organism's resistance to decompression disturbances incident to repeated decompression effects is based on improvement of the defense reactions of the respiration and blood circulation that are evoked by the aeroembolic process. These reactions reduce to an increase in pulmonary ventilation, amplification of heart action and increase in the intensity of blood circulation; they intensify the process of liquidation of gas bubbles that have already been formed and restrict the development of new ones. A vital and perhaps even the determinative part in the mechanism of development of these reactions is played by shifts in the gaseous composition of the arterial blood that arise as a result of the plugging up of the pulmonary capillaries with air emboli.

The organism's ability to increase its resistance to decompression disturbances incident to repeated decompression effects must be taken into account and utilized in practice.

PART III

ALTITUDE DECOMPRESSION DISORDERS

CHAPTER 13

HISTORY OF THE STUDY OF ALTITUDE DECOMPRESSION DISORDERS

The history of the study of altitude decompression disorders/244 is one of the clearest examples of how the formulation and development of scientific problems is determined by the requirements of everyday life. To be historically correct, the source and origin of the entire problem of decompression disorders as a whole ought to be connected with the "altitude" side of this problem and ought to be attributed to the three-hundred-year-old investigations of Robert Boyle [1670].

As has already been pointed out, R. Boyle was the first to observe bubbles in the fluid media of the organism incident to the effect of considerable degrees of rarefaction of the atmosphere. Not to be excluded is the possibility that in R. Boyle's observations, the phenomena of the boiling of fluids incident to critical degrees of rarefaction were combined with the phenomena of the transition of gases from the dissolved state to the free state. In that case, this author must be regarded as the discoverer of both these categories of phenomena which occur in the organism incident to rarefaction and which, from the physical point of view, at the same time have much in common. Be that as it may, in his studies, R. Boyle directly pointed to gas formation in the organism incident to rarefaction as a possible cause of fatal functional disturbances and, consequently, was the first to proclaim the existence of the problem of decompression disorders. At that time, however, there existed no questions of vital importance proceeding from man's practical activity which might have aroused interest in this problem, and it did not attract the attention of researchers or undergo development. Added in the 18th century to the pronouncements and conclusions of R. Boyle was the report by Van Musshenbrok having the same content, which did not attract any wide interest, either.

In the 19th century, two hundred years after the studies of R. Boyle, Hoppe [1857] wrote persuasively about the phenomenon of gas formation in the organism and, in particular, in the blood incident to the effect of a rarefied atmosphere. It is remarkable that his pronouncements belong to that period /245 when the practice of caisson work was bringing out the problem of decompression disorders in all their acuity, was attracting wide interest to itself and was giving a powerful stimulus to

its scientific treatment. But again, inasmuch as the effect of considerable degrees of rarefaction of the atmosphere on man was of no practical significance, allusions to the possibility of altitude decompression disorders went unnoticed and were not linked up in any way with the abundant material about decompression disturbances incident to transition from increased pressure to normal pressure. This material and this sphere of man's practical activity serve as the basis for valid formulation and intensive scientific treatment of the problem of decompression disorders, but the conceptual sources of this problem, which are connected with the effect of a rarefied atmosphere, underwent, as before, no development for a long time. P. Bert, who subjected himself repeatedly to the effect of rarefaction corresponding to altitudes of up to 8000-8800 m, apparently experienced no altitude decompression disorders; in any case, in his studies and experimental reports we find no description of the corresponding symptoms.

Haldane theoretically foresaw the possibility of altitude decompression disturbances when the pressure is reduced to 300 mmHg and less (altitudes in excess of 7 km). He did not check this possibility experimentally; at the same time, Haldane in his studies referred to a description given in 1906 by von Shretter of the symptoms he experienced that resembled those of "caisson disease" incident to rarefaction of the atmosphere corresponding to an altitude of 9,000 m. Haldane pointed out that von Shretter was apparently the first man to test on himself and describe the symptoms of altitude decompression disorders, but was unable to give them the correct interpretation. Fulton [1948], in referring to the pronouncements made in 1917 by John Henderson, called the latter the first physiologist to acknowledge the possibility that decompression disorders may arise at an altitude. To judge from Fulton's references, however, these pronouncements were abstract in nature and were not based on any experimental data or personal impressions.

In 1930 Jongbloed published a cogent report about the development in him and his colleague of pronounced joint pains incident to rarefaction corresponding to an altitude of 13,000 m, stressing the similarity of the symptoms they experienced and the manifestations of "caisson disease." Still more cogent results of self-experiments with the development of altitude pains were published in 1931 by Barcroft, Douglas, Kendal and Margaria. These researchers determined the maximum altitudes at which physical efficiency does not yet suffer when oxygen is breathed. In a rarefied atmosphere in a pressure chamber, they performed physical work (stepping up on a 32-cm-high box 15 times a minute for one hour) and came across the fact of regular development of pronounced pains in the knee joints and in the adjacent muscles. The authors refrain from any definite interpretation of the cause of the pains they experienced, /246

stressing at the same time the necessity of elucidating this important phenomenon. As soon as researchers started becoming familiar with the effects caused by a low pressure, and once again in the course of self-experiments, in addition to altitude pains, considerably more serious consequences of the effect of a rarefied atmosphere were also brought to light.

Boothby and Lovelace [1938] reported on a case of paralysis of the lower part of the body that developed rapidly in their colleague Dr. Hayme, who was the examinee in a pressure chamber at an altitude of 10,500 m, breathed pure oxygen and felt no indications of hypoxia. Upon returning to normal pressure, these threatening paralytic manifestations vanished without a trace. But none of the above cases or individual data about the effect of a rarefied atmosphere on the organism was yet clearly classified at that time as an expression of altitude decompression disorders. Their nature did not seem clear or firmly established.

All of these data anticipated the approaching practical necessity of studying altitude decompression disorders, but in the absence of a powerful practical stimulus for interpreting this side of the problem, the above investigations did not receive any extensive development. At the end of the Thirties, as a result of the observations presented above and individual data from flying practice, only a general impression was accumulating that, besides the relatively well-studied danger of hypoxia, against which a defense had already been worked out in the form of oxygen masks, high altitudes also harbor an important additional threat to man in the form of muscular and joint pains and more serious functional disorders of unexplained etiology.

The practical stimulus for intensive study of these phenomena, a stimulus which called to life and development this historically first sign of the entire problem of decompression disorders, came together with the start of the Second World War. It became eminently clear from the combat operations of aviation that their success at that time was completely determined by the speed, maneuverability and, most important, altitude ceiling of flights. Toward the end of the First World War, the idea of pressurized cabins was still going through its first stage of testing, and solid physiological grounds had to be worked out. In military aviation, flights in open cabins, which were directed at achieving ever greater altitudes, as well as altitude tests by flying personnel in pressure chambers, which were being widely put into practice, immediately started to yield extensive practical material on the development of altitude muscular and joint pains. Life demanded an immediate solution to the question of their nature and prophylaxis. These vitally/247 important requirements were the powerful stimulus that was needed for scientific working out and development of that side of the problem of decompression disorders which is connected

with the effect of a rarefied atmosphere on the organism. Under the influence of this stimulus, extensive investigations began to be undertaken abroad -- in particular, in Germany and in the United States of America (which were mentioned above) -- as well as here in the Soviet Union. To this period belongs the intensive experimental activity of the prominent representatives of domestic aviation medicine -- V. V. Strel'tsov, A. A. Sergeyev, D. Ya. Rozenbloom, I. M. Khazen, as well as the many-sided and fruitful investigations of M. P. Brestkin and his collaborators -- V. A. Skrypin, V. V. Levashov, Yu. L. Komendantov, V. V. Portugalov, V. A. Vinokurov and others, which were directed at solution of the most acute and vitally important questions of aviation physiology.

It should be said that the stormy development of aviation, coinciding as it did with the years of the Second World War, in addition to the question of the effect on the organism of rarefaction and development of altitude decompression disorders, at once brought out a great number of other interrelated problems of great urgency -- such as the creation of pressurized cabins, altitude equipment, instruments for breathing under overpressure and the physiological basis and working out of detection methods. In the solution of these problems that brooked no delay, a particular significance and special place was occupied by the activity of the mentioned team headed by M. P. Brestkin, which was conducted under the general guidance of L. A. Orbeli. The investigations carried out in this period by M. P. Brestkin and his collaborators were distinguished, if one may put it that way, by rare operationality; they were of necessity connected with elements of no small risk but, owing to the broad general physiological erudition of the leaders, their profound knowledge in the region of the special problems being worked out and the first methodological approaches to solution of the problems that had arisen, they passed practically without harm or injury to the health of the examinees. These investigations combined scientific profundity with practical single-mindedness and efficiency. Thus, under real conditions, Vinokurov, Levashov and Khromushkin conducted investigations devoted to the effect of overloads on the organism incident to changes of attitude of high-speed aircraft, which permitted flying personnel to be correctly oriented in evaluating the subjective sensations that arise under these conditions; M. P. Brestkin with his collaborators in a very short time solved the problem of ejection, and this only possible means of saving the pilots of high-speed aircraft in emergency situations was put into practice. To this same period belong V. A. Skrypin's self-experiments, unparalleled in their daring and directed at studying the effects on the organism of extreme altitudes, the role of carbon dioxide under these conditions and the efficiency of breathing oxygen under an over-/248
pressure in the lungs.

As far as the effect on the organism of drops in pressure, acute degrees of rarefaction and decompression disorders is concerned, this question was intertwined with the problem of creating pressurized cabins and acquired special urgency. Its solution was complicated by the fact that some researchers, in automatically transferring Haldane's stand about the maximum permissible coefficient of supersaturation to flying practice, asserted that incident to rapid rarefaction of the atmosphere down to 1/3 of its normal pressure, man was threatened with fatal decompression disorders. These assertions ensued to a considerable extent from incorrect interpretation by individual researchers of the results of experiments on goats with the effect of the above-mentioned pressure drops. These animals, as is well-known, very clearly display the painful decompression symptoms involving the extremities (bends symptoms) upon return from under increased pressures. They were widely and successfully used by Haldane in his experiments directed at working out prophylactic decompression methods.

It was natural that some researchers should decide to use goats for studying altitude decompression disorders, too. Here were obtained results which entailed the above-mentioned incorrect conclusions. Incident to rapid drops in pressure down to 1/3 of the normal, goats very often developed serious and even fatal disorders, section of killed animals disclosing bubbles in the sanguiferous vessels. This picture was interpreted as the consequence of severe, truly decompression, disorders. Actually, however, it turned out that the essence of the above phenomena caused by drops in pressure consisted of something else. Goats are distinguished by the peculiar structure of their digestive tract and very long intestine, which always contains a great amount of gases. The expansion of these gases due to a rapid drop in external pressure leads to acute reflex effects on the cardiovascular activity and respiration, and in a number of cases cause ruptures of the walls of the gastrointestinal tract, with the ingress of air into the sanguiferous system. Hence was created the impression of acutely pronounced, truly decompression, aeroembolic phenomena and ensued conclusions about the critical coefficient 3 that itself determined the organism's limit of endurance of drops in pressure due to rarefaction of the atmosphere. The fatal coefficient 3 forged the thinking of designers that was directed at creating pressurized cabins and selecting pressure conditions in them.

Correct solution of the question of the effect on the organism of abrupt drops in pressure hinged upon the principles of design of pressurized cabins and advances in altitude aviation. This question, too, was clarified in a very short time by M. P. Brestkin and his collaborators (Bondarenko, Zhironkin, Ivanov, Shistovskiy). /249

Investigations carried out on people, including some of the authors themselves, showed that man can painlessly endure sudden second-long drops in pressure not only down to 1/3, but even down to 1/7-1/8 of its usual value. This conclusion concerned the direct effect of the drop as such, i.e., in other words, it referred to its mechanical effect and its immediate consequences. At the same time, it was shown in these investigations that incident to considerable degress of rarefaction of the atmosphere corresponding to altitudes of more than 9 km, some time after the drop people generally develop true decompression disorders connected with formation of gas bubbles in the organism and manifesting themselves in itching of the skin, muscular, joint and thoracic pains, deterioration of general "self-sensation" and sometimes even in the development of precollapsoid conditions.

It became clear from the results of these investigations and analysis of previous data in the literature that still another mistake had been made in interpreting the results of the pressure-drop experiments on goats. This mistake was that it was incorrect to assume such a rapid, almost instantaneous, formation of gas bubbles in the organism as a result of supersaturation of its tissues and blood with an inert gas. This process, as was shown by the above-mentioned data and numerous subsequent direct results of studying it, always requires a certain time. This time is the shorter, the higher the degree of supersaturation of the tissues; but nevertheless, even in cases of exceptionally rapid decompression from under increased pressures, when this degree of supersaturation is very high, formation of gas bubbles goes on not instantaneously, but is always, under conditions of rarefaction, more or less dragged out in time.

Returning to the history of the development of the "altitude" side of the problem of decompression disorders, it must be said that as a result of the above-mentioned intensive investigations stimulated by the development of aviation that were carried out in the Soviet Union as well as abroad, toward the end of the Forties of the present century the conditions under which altitude decompression disorders develop and, most important, their nature and essence, had, on the whole, been pretty much clarified. Alongside of physiological analysis of altitude decompression disorders, the question of their prophylaxis in fliers had been solved. This was furthered by the perfecting of oxygen equipment and the working out of pressurized cabins. As a result, recommendations about the prophylaxis of altitude decompression disturbances by means of comparatively short-lived breathing of oxygen, which caused a corresponding desaturation of the organism from nitrogen, were put into practice, recommendations that satisfied the requirements of the aviation of that time.

Experimental investigations carried out on people in /250
pressure chambers (including those conducted in the pressure laboratory of the Military Medical Academy under the guidance of M. P. Brestkin with the participation of the author of the present book) convinced us of the efficiency of such prophylactic measures. In these experiments, the staying times of the examinees at high altitudes were generally limited to no more than 1-1.5 hours, and physical loads were usually not used. Under these conditions, after first breathing pure oxygen before ascent for one hour and even 30-40 minutes, signs of altitude decompression disorders did not, as a rule, appear. The impression of the complete effectiveness of the given prophylactic method was reinforced also by experiments in which the direct goals of studying strictly decompression disorders were not pursued. These experiments included investigations on people (to a considerable extent in the form of self-experiments) and experiments on animals involving study of the physiological effects of breathing oxygen under an overpressure [Botvinnikov, Ginsberg, Graminitskiy et al, 1955]; and experiments involving study of the comparative role played by the pressure and content of oxygen in the blood in the development of hypoxemic phenomena [Vinogradov, Graminitskiy et al, 1958].[1]

In all of these investigations, including those conducted on animals, the participants and the experimentors were obliged to "rise" in the pressure chamber to "altitudes" on the order of 10-13 km with a stay there of up to 1.5-2 hours. In conformity with the established rules, before the ascent comparatively short-lived (up to one hour) desaturation from nitrogen was effected by means of breathing oxygen, and all these experiments generally took a favorable course, i.e., with no pronounced symptoms of altitude decompression disorders. Only in individual cases, at altitudes that were the maximum permissible ones from the standpoint of oxygen provision (12-13 km), did the experimentors note the appearance of itching and mild pains, which did not interfere with the accomplishment of the planned experimental program. In general, all of this experimental practice, on which I have permitted myself to dwell in greater detail as a direct participant, as well as all of the host of analogous materials obtained in different foreign and domestic laboratories, and flying practice, too, engendered and strengthened the conviction that relatively short-lived desaturation from nitrogen by means of breathing oxygen pretty reliably prevents the development of altitude decompression disorders. In conformity with this, inasmuch as the practical side of the question

[1] The studies by the above-mentioned authors were carried out at the end of the Forties and the beginning of the Fifties, but were published considerably later.

as applied to the requirements of the aviation of that time /251
seemed to be satisfactorily solved, the wave of experimental investigations devoted to analysis of "altitude" decompression disorders that had risen in preceding years, also subsided. And although a great number of physiological questions concerning the distinctive features of altitude decompression disturbances remained unsolved, this side of the problem generally began to be regarded as not so acute or urgent.

A powerful new practical stimulus for working it out was provided by the development of astronautics. As has already been mentioned in the introduction to the present book, the practice of space flights introduced two new conditions concerning the effect on man of the low surrounding pressure existing in space suits. The first was the need for a man to stay under a low pressure for a long time, perhaps even many hours, and the second was the inevitability of simultaneously performing more or less hard physical work. These two conditions, as has been pointed out, placed the "altitude" side of the problem of decompression disorders, and even the entire problem of decompression disorders as a whole, in a new light, raised a number of interesting and unexpected questions, brought out the unreliability of the prophylactic measures adopted in aviation practice and showed the necessity of putting the rational and effective protection of astronauts against decompression disturbances on a more solid scientific basis. Here there is no need to dwell in greater detail on all of these circumstances inasmuch as their detailed analysis with the presentation and discussion of the pertinent experimental material constitutes the content of the following chapters.

CHAPTER 14

DECOMPRESSION DISORDERS INCIDENT TO TRANSITION FROM NORMAL PRESSURE TO DIFFERENT DEGREES OF ATMOSPHERIC RAREFACTION

Altitude decompression disorders in man incident to the action of different degrees of atmospheric rarefaction without preliminary desaturation

There can be no doubt that the study of how altitude decompression disorders develop incident to the action on man of different degrees of atmospheric rarefaction without preliminary desaturation is quite indispensible if we wish to

obtain initial data on the basis of which effective measures /252
of their prophylaxis as applied to space flight conditions are to be worked out. In the present chapter, in conformity with the procedure that was followed in preceding sections of the book, an account is given mainly of the experimental data that the author and his colleagues obtained in connection with the above question. In the present section of the book, I have not set myself the task of reporting on the results of corresponding experiments by different researchers that are to be found in the literature, so that in those cases where the results we obtained coincide with the data in the literature, I shall confine myself to short indications to this effect, thereby stressing that the adduced facts are typical; on the other hand, special attention will be paid to those cases where contradictions arise or where our results are new as compared with those in the literature.

We are justified in beginning our description of the decompression disorders that arise in people incident to ascents to high altitudes [2] without preliminary desaturation with one of the early studies conducted by the author and his colleagues [Brestkin, A. P. et al 1955], which was carried out in 1952 without any connection with the requirements of astronautics and, consequently, without taking into account the two specific above-mentioned conditions (length of action of the rarefaction and physical load), which have determined the new aspects of this whole problem. It is profitable to do this because, in the first place, it will be possible to compare with the results of the study in question the data of subsequent investigations in which the staying times at an altitude increased and a physical load was introduced and, in the second place, the study in question was conducted in the form of self-experiments with detailed analysis and strict evaluation of all subjective sensations -- the chief criteria for the development of decompression disorders.

The investigations were carried out in the form of self-experiments on five examinees -- practically completely healthy men ranging in age from 30 to 40 years. The examinees were subjected to the effect of a rarefied atmosphere in a large pressure chamber (53 m^3 in size); in the main experiments, the rarefaction amounted to an altitude of 10.5-11 km, while in supplementary experiments set up for control purposes, it

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[2] Here and in what follows, in speaking about investigations in pressure chambers, I shall permit myself to use the expression "ascents to an altitude" without quotation marks inasmuch as they are generally accepted as a designation for the effect of the corresponding degrees of rarefaction.

of the thermocouples from the mirror galvanometer; the third studied conditioned reflexes.

Temperatures were continuously measured throughout the entire experiment (the indications of each thermocouple were recorded for 5 min); the blood pressure and the respiratory and pulse rate were measured at 15-minute intervals twice before ascent, four times at the altitude and twice after descent. The experiments were conducted twice a week, in the first half of the day.

Besides the main task of the experiments -- study of the development of decompression disturbances in people incident to the action of a rarefied atmosphere -- two other questions were studied: the effect of a water and water-salt load and the effect of temperature conditions on the development of decompression disorders. /254

The first question was raised in connection with the data obtained by A. P. Brestkin on the lesser ability of hydrated tissues to retain dissolved nitrogen in a state of supersaturation as compared with tissues poorer in water. The basis for raising the second question -- on the significance of temperature conditions for the development of decompression disorders -- was diving practice data indicating that a low ambient temperature predisposes to development of decompression disturbances.

In the experiments with a water load, the examinees drank 1.5 ℓ water at room temperature 30-60 min before ascent; in the experiments with a water-salt load, each examinee ate 200-250 g herring and 100-150 g bread before drinking the water. The sodium chloride content in the herring amounted to about 10%. Thus, together with the herring, 20-25 g salt was introduced into the organism. In both versions of the experiments, besides recording all of the above-mentioned indices, the diuresis was determined. After drinking the water, the examinees emptied their urinary bladders every 20 or 30 min.

In the experiments involving study of the effect of temperature conditions on the development of decompression disturbances at an altitude, the examinees did not wear their ordinary clothing in the chamber, but fur flying suits, caps with ear flaps and felt boots. This hampered the organism's heat emission. Control experiments with ascents to an altitude of 5-5.5 km were undertaken in connection with the fact that at an altitude of 10.5-11 km (in the main experiments), in spite of breathing pure oxygen, some hypoxemia was observed. It was necessary to elucidate to what extent it was responsible for those functional shifts which were recorded in the examinees during their stay at an altitude of 10.5-11 km, where decompression disturbances developed.

amounted to an altitude of 5-5.5 km. In the first case, the examinees breathed oxygen out of KP-14 apparatus from the start of the ascent, and in the second case, air. The rate of ascent and descent was on the average equal to 2 km per 1 min. The staying time at the altitude amounted to 1 hour; if any one of the examinees developed intolerable joint pains or, independently of them, his general condition deteriorated, the descent was begun earlier. /253

In the experiments, the nature of the decompression disorder symptoms and the frequency of their development was studied, the upper nervous activity of the examinees was investigated and the cutaneous, axillary and rectal temperatures, blood pressure and pulse and respiratory rate were measured. All outwardly noticeable decompression disturbance symptoms were carefully recorded by the experimentor who was observing the examinees. The latter, moreover, wrote down all of their sensations in detail.

Upper nervous activity was studied by the method of motor reflexes with verbal confirmation based on preliminary instructions. Temperatures were measured in the experiments with the aid of homemade thermocouples and a mirror galvanometer. The ends of the thermocouple, with junctions that sensed the examinees' temperature, were introduced into the pressure-chamber through a connecting pipe with a gasket. A Dewar vessel with "cold" junctions and the galvanometer were placed inside the chamber. The temperature was measured in the rectum (to a depth of 7 cm), in the armpit and at seven points of the surface of the skin: the thorax, the back, the abdomen and the extremities. Measurement accuracy for absolute temperature values amounted to 0.1°, and for its relative changes, to 0.05°.

The arterial blood pressure was measured by the usual method of Korotkov. The examinees in the chamber measured one another's blood pressure and pulse rate. The respiratory rate was read off from the indicator of the oxygen respiratory apparatus.

There were two (in some cases three) men inside the chamber in each experiment. Conditioned reflexes were investigated in one of them. Each of the men in the chamber, in addition to recording his own sensations, had a definite obligation in conducting the experiment: he had to keep track of his partner's condition, determine his pulse and respiration, measure his blood pressure. One of the participants in the "ascent" was appointed responsible experimentor in the chamber: he kept contact with the participants of the experiment outside the chamber. From outside the chamber, three experimentors worked. One of them stood at the portholes throughout the entire experiment and continuously kept track of the behavior and condition of the examinees; another experimentor recorded the indications

In all, 63 experiments were set up at an altitude of 10.5-11 km (of which 39 were without any additional effects, four were with a water load, 14 were with a water-salt load and eight were with warm clothing) and eight experiments, at altitudes of 5 and 5.5 km.[3]

Let us here dwell on the general results of the experiments, leaving the data on upper nervous activity for the concluding section of the present chapter, where to these data will be added the results of investigations that our team carried out in /255 this area recently.

Rapid rarefaction of the air corresponding to an altitude of 10.5-11 km in the great majority of cases caused the examinees to develop decompression disorder symptoms of one sort or another: itching of the skin, marmoreal skin, pains in joints and muscles, etc.

Itching of the skin is the earliest and most frequent symptom. Usually this sensation arose in the first 10-15 min of the stay at the altitude. Most often, the itching was localized on the skin of the inner surfaces of the forearms, the stomach and the back, then moved on to the dorsal surface of the forearms and hands and to the cutaneous integuments of the shoulders, thighs and shinbones. Not seldom, together with the itching, a tingling sensation arose on the skin of different regions. Both the itching sensation and the tingling sensation were transient for individual regions of the skin: having arisen in some part or another, they disappeared from there and appeared in another part, then reappeared in the first part, etc.

In approximately 1/3 of the cases, more serious decompression symptoms were noted, namely: pains in the joints and muscles that arose in different examinees with different frequencies and intensities. As a rule, the pains appeared 15-30 min after reaching the altitude against a background of already existant itching. Their localization was rather typical: most often, they arose in the joints of the extremities, especially the radiocarpal, elbow and shoulder joints; more rarely, in the talocalcaneal and knee joints. Intensification of the pains was accompanied by their spreading to the adjacent muscles and along the course of the nerve trunks. Besides the joints and muscles of the extremities, painful sensations sometimes arose in the region of the breastbone. Finally, in a number of experiments, the examinees developed difficulty in breathing, which was accompanied by a dry uncontrollable cough, and some-

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[3] We took the ascent to an altitude of one examinee per experiment.

times sweating. These symptoms were always accompanied by deterioration of the examinee's general condition, for which reason we had to break off the experiment and resort to descent. Upon descending to an altitude of 8,000-6,000 m, the cough and the sweating, as well as the other decompression symptoms, disappeared.

When the examinees developed subjective sensations -- itching, pains and other mainfestations -- the observer at the porthole, as well as the experimentor inside the chamber, recorded corresponding external reactions: scratching of the skin, its maculation ("marbling"), unnatural position of the extremities in whose joints there were pains, sharp curtailment of their movement, etc.

The above-mentioned subjective sensations and visible changes in the examinees' condition were generally accompanied by shifts recorded by us in the objective indices of upper nervous activity, which will be the subject of discussion below, changes in function of the cardiovascular system and respiration, as well as in internal and peripheral body temperature. A very characteristic phenomenon was an increase in skin temperature coinciding with the development of cutaneous itching. The increase in temperature upon appearance of itching was most pronounced on the cutaneous integuments of the forearms, where the itching sensation arose in the examinees most frequently and was most intensive (Fig. 70). /256

In a number of cases, the development of very pronounced decompression disorders at the altitude was accompanied by an increase in rectal temperature in the range from 0.15 to 0,2°C. This fact is in agreement with observations of physicians supervising caisson operations, who more than once have noted an increase in body temperature in divers and caisson workers incident to the development of decompression disorders in them. We must suppose that this increase in body temperature is also a peculiar neuroreflex reaction accompanying decompression disturbances. Its presence in our experiments is of particular interest in connection with the fact that it developed under conditions of some hypoxemia, which, as is well-known, in itself causes a drop in internal body temperature.

Upon ascending to an altitude of 10,500 and 11,000 m, the pulse and blood pressure of the examinees changed identically in all experiments: the frequency of the heartbeat increased inconsiderably, and the level of the maximum and minimum blood pressures increased somewhat. Usually, soon after reaching the altitude, these indices became fixed at a certain somewhat increased level and subsequently did not change essentially, if the examinees did not develop decompression pains. Generally noted with the development of pains was an

additional increase in the pulse rate, and the maximum and minimum blood pressure levels changed: in some cases it increased, in others it decreased. Former cases arose more frequently for low- and medium-intensity pains, and the latter, for especially acute painful sensations. The degree of change in the blood pressure for pains as compared with its level before the development of pains usually did not exceed 10-15 mmHg.

Incident to the development of decompression disorders, the respiration also changed. At first there was a small increase in its rate and depth, and then, with the development of altitude pains, it became arrhythmical and nonuniform in depth.

The general results of the main experiments involving ascents to an altitude with no additional effects, as well as the experiments with a water and water-salt load and with restriction of heat emission, are shown in Table 13. In conformity with what has been said, the data of Table 13 are convincing evidence that incident to the action of rarefaction corresponding to altitudes of 10.5-11 km, in the overwhelming majority of cases man develops specific decompression symptoms of one kind or another, i.e., decompression phenomena develop in the organism.

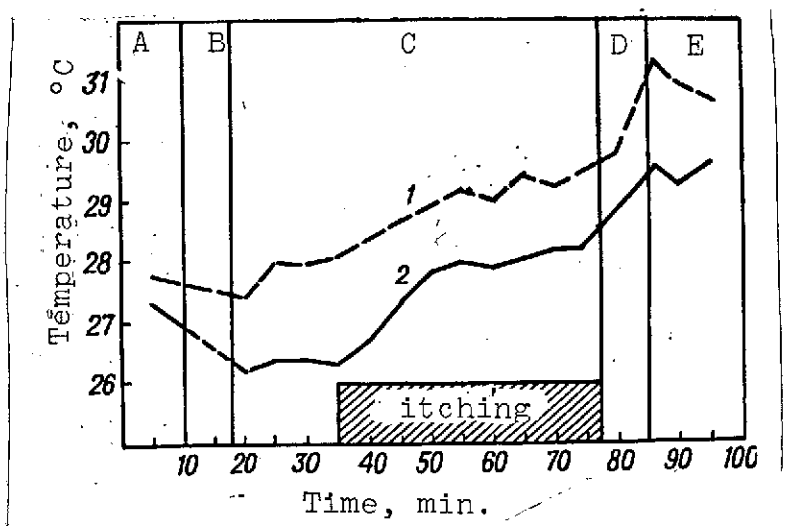

Fig. 70. Changes in skin temperature of examinee in the region of the right (1) and left (2) radiocarpal joints incident to the development of decompression itching of the skin at an altitude of 11,000 m. A -- before ascent; B -- ascent; C -- stay at an altitude of 11,000 m; D -- descent; E -- after descent.

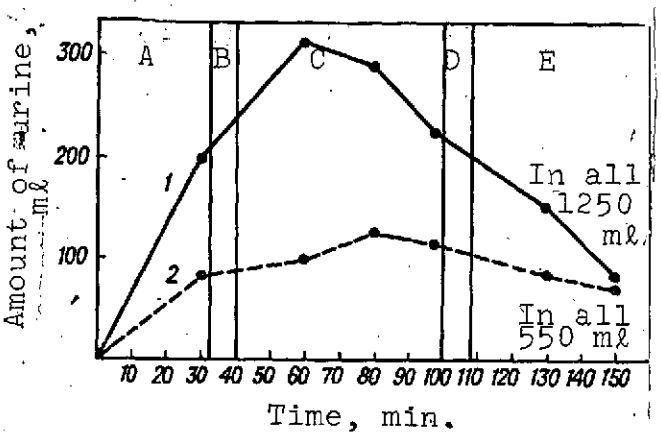

Fig. 71. Diuresis of examinee after water and water-salt loads at an altitude of 11,000 m.
1 -- for water loads;
2 -- for water-salt loads;
A -- before ascent;
B -- ascent; C -- stay at the altitude; D -- descent;
E -- after descent.

A water load, as the experiment showed, exerts no substantial effect on the development of decompression disorders, whereas with a water-salt load, they develop more frequently and prove to be more pronounced. Clearly, this is connected with the increased hydration of the organism's tissues in experiments with a water-salt load. The data shown in Table 14 and Fig. 71 on the extent and dynamics of diuresis in the experiments under consideration point to a considerable retention of the water introduced into the organism when salt is taken at the same time. /258

The experiments with curtailment of heat emission, on the other hand, are distinguished by their more favorable results. Decompression symptoms appeared in these experiments more seldom and were weaker. This effect, we must suppose, is connected with an increase in the blood circulation in the skin and subcutaneous cellular tissue, which is

TABLE 13. FREQUENCY OF APPEARANCE OF DECOMPRESSION SYMPTOMS IN EXAMINEES AT ALTITUDES OF 10,500-11,000 m UNDER DIFFERENT EXPERIMENTAL CONDITIONS

Conditions	Number of experiments	Absence of symptoms	Symptoms		
			Itching skin	Joint and muscular pains	Substernal pains, cough, sweating
Without additional effects	39	2	37	16	5
With water load	4	1	2	1	-
With water-salt load	14	1	4	9	2
With curtailed heat emission	8	4	2	2	-

indicated by the considerable increase (by 3-5°) in the average level of the skin temperature and, probably, also by the increase in intensity of the general blood flow. The adipose subcutaneous cellular tissue is one of the main depositories of the nitrogen dissolved in the organism; its more rapid desatura- /259
tion not only prevents the appearance of local symptoms connected with gas formation in the subcutaneous vessels (itching, marbling of the skin), but reduces the danger of pronounced general aeroembolic phenomena developing.

TABLE 14. EXTENT OF DIURESIS IN EXAMINEES AFTER WATER AND WATER-SALT LOADS UNDER ORDINARY CONDITIONS AND AT ALTITUDES OF 10,500-11,000 m.

	Urine liberated at 2 h 30 min after			
	Water load		Water-salt load	
Examinee	Under ordinary conditions	At the altitude	Under ordinary conditions	At the altitude
1	1325	1195	525	555
2	1370	1250	490	550
3	975	1125	575	625
4	1110	970	350	450

It should be noted that the just-mentioned data agree with the indications in the literature about the positive effect of heat and the negative, provocative effect of cold on the development of decompression disorders [Yakobson, 1950; Fulton, 1951; Aver'yanov, 1960, 1962].

The data on the negative significance of increased hydration of the tissues for the development of altitude decompression disturbances is new and, we must suppose, deserves attention in spite of the fact that from the quantitative point of view they are incontestibly insufficient for categorical conclusions.

As far as the general results of these investigations are concerned, when compared with the data in the literature they are rather typical and, to judge from everything, faithfully reflect the nature and frequency of the decompression disorders that develop incident to ascents to higher altitudes without preliminary desaturation under the condition of relative muscular repose during the stay in the rarefied atmosphere.

In evaluating these data and comparing them with the data encountered in the literature, the only circumstance that must without fail be taken into account is what and how pronounced are the symptoms in terms of which different authors determine the frequency of development of altitude decompression disturbances. That is why the figures adduced in individual studies (usually, percentages) as reflecting the frequency of appearance of decompression disorders can vary sharply. As the criterion for the frequency of development of altitude decompression disorders, as well as decompression disorders in general, authors usually use muscular and joint pains that are clearly perceived by the examinees. This is generally accepted and incontestibly justified. In all of our investigations, too, this was used as the main and determinative symptom.

But it is absolutely necessary to pay attention also to various milder (as it is customary to regard them) and earlier manifestations of decompression disorders, in particular such a strikingly outstanding symptom of them as itching of the skin. Especially important and absolutely obligatory are ascertainment and evaluation of different early symptoms of altitude decompression disorders and, in particular, itching of the skin incident to ascents to an altitude without preliminary desaturation, when there is a clear and real danger of aeroembolic phenomena. It can be assumed that the overwhelming majority of researchers are unanimous in interpreting the cause of postdecompression itching of the skin as a consequence of gas-bubble formation in the very fine venous vessels of the subcutaneous cellular tissue. The results of our experiments /260 argue in favor of this interpretation.

The development of itching in our self-experiments, which was generally preceded by the appearance of painful symptoms, was usually accompanied by appreciable changes in the organism's general condition. In order to judge of changes in the organism's general condition, the self-experiment is an irreplaceable form of investigation inasmuch as it affords the possibility of carefully analyzing subjective sensations and comparing them with objectively recorded physiological shifts. At the same time, subjective sensations prove in a number of cases to be a finer criterion for changes in the organism's general condition than the results of objective investigations. Owing to the rare ability of the nervous system to put into equilibrium conditions of its activity that have changed, and, in so doing, to maintain its full-fledged functioning, some of the organism's reflex responses to external stimuli may remain unchanged, while the man is clearly aware of a condition of general ill-being.

Such sensations generally arise incident to the development of decompression itching. This symptom, which is harmless in itself, entails no great unpleasantness but, as a rule, simultaneously with its development the general self-feeling

deteriorates perceptibly. Usually it is not possible to reduce this deterioration to any concrete perceptions, and sometimes all that can be noticed is that it is becoming more difficult to breathe and that there is not enough air; but, independently of this, incident to the development of itching of the skin, it is almost always possible to note for a certainty that one's condition no more than 5-10 min ago was different and has now taken a turn for the worse. As a rule, with intensification of the itching, the deterioration in the general condition progresses; the development of painful symptoms aggravates this. The general condition deteriorates especially noticeably and especially rapidly in those cases where a cough, a feeling of tightening in the chest and substernal pains develop. At the same time, a cold sweat develops, dizziness sets in and, not infrequently, a clearly perceptible threat of fainting arises, which compels immediate descent to be requested. The normalization of the general condition incident to descent takes place rapidly and is perceived extremely clearly. As a rule, as early as an altitude of 6-5 km, all unpleasant sensations pass completely /one after another without leaving a trace, which is accompanied by clearly perceptible euphoria.

None of this picture can be easily explained in any other way than by development of an aeroembolic process leading to a plugging up of part of the pulmonary capillaries and intensifying the hypoxemia that arises at an altitude of 10.5, and all the more so, 11 km in spite of the breathing of pure oxygen. Clearly, it is precisely by this intensification of hypoxemic phenomena that one must explain the circumstance that at an altitude even the development of the very first and weakest decompression symptoms (slight itching of the skin) very often coincides with a clear deterioration of self-feeling, while /261 the appearance of the same symptoms after emerging from a depth, when the organism does not experience hypoxemia before the start of aeroembolic phenomena, is not accompanied by any appreciable change in the organism's general condition.

Thus, there are grounds for supposing that incident to the action on the organism of acute degrees of atmospheric rarefaction without preliminary desaturation, as well as incident to ascents from a depth, the first and most important link in the development of decompression disorders is the aeroembolic process, which has been analyzed in detail in the preceding section of the present book.

In this connection, we must briefly treat the question of the change in man's resistance to decompression disorders incident to repeated actions of a highly rarefied atmosphere without preliminary desaturation. In the investigation that we have been examining, we systematically employed effects of one and the same intensity, regardless of whether pronounced

decompression disorders did or did not develop in the examinees in the course of the experiments; in other words, we did not set ourselves the goal of creating for each examinee the necessary conditions for increasing his resistance to decompression disorders. It is in consequence of this that the changes in the examinees' resistance in the course of the study proved to be different.

Those examinees (two men) in which during the course of the experiments pronounced decompression disturbances developed repeatedly, showed an increase in resistance. It is significant that at the same time, a change in the examinees' condition was noted (in particular, a change in upper nervous activity) even before the start of the ascent. It is most likely that the shifts in question were the result of negative conditioned reflexes perfected in preceding experiments on the basis of significant decompression disorders that it was difficult to endure subjectively. The examinees testified that they began to fear the development of these disorders in the next experiment. In two other examinees, who, in a number of experiments, did not experience serious decompression disturbances following one another, the resistance to them in the course of the study did not change significantly, while in one examinee, it increased considerably. Whence it can be assumed that for decompression disorders that arise upon transition to higher altitudes without desaturation, those general conditions remain valid which determine the changes in the organism's resistance to decompression effects that were examined earlier as applied to ascents from a depth. When the conditions that are conducive to the conditioning of the organism are adhered to -- gradual intensification of the effects with their temporary weakening after cases of very pronounced functional disorders -- the organism's resistance to altitude decompression effects can probably be increased.

Let us now turn to recent experimental data on altitude /262
decompression disorders in connection with the requirements of astronautics. Here again I must caution the reader that the purpose of the following account will be, first of all, to present experimental data obtained by the author and the team he led, and does not aspire to quote and analyze in full those data in the literature dealing with separate questions of altitude decompression disorders which will not shake the validity of the conclusions drawn below. Inasmuch as the obtained experimental data are in good part original and lead to a really alternative solution of the problem of decompression disorder prophylaxis as applied to the conditions of domestic astronautics, their publication, it must be supposed, affording the possibility, as it does, of their extensive discussion and interpretation by specialists in the given field, is justified and will further both correct solution of the problems posed

by space flight and theoretical development of the problem of altitude decompression disorders.

It must be said that those systematic investigations of the problem of altitude decompression disorders which have been carried on in recent years by myself and my colleagues are a continuation and development of the work begun by A. G. Kuznetsov, I. P. Poleschuk et al. A. G. Kuznetsov, I. P. Poleschuk et al, many of whom are now working in our team, carried out the first stage of these investigations. It was important, in the first place, because it included the organizational difficulties connected with the start of any great work, and, in the second place, because -- and this is the main point -- this stage disclosed the insufficient effectiveness of desaturation from nitrogen by means of breathing oxygen for decompression disorder prophylaxis as applied to space flight conditions and thereby placed the problem of altitude decompression disorders in a new light.

The investigations of A. G. Kuznetsov et al, as well as all of our investigations, were conducted with observance of those two conditions which ensue from the practice of space flights and to which we have already repeatedly referred, namely, a man's rather long stay (usually lasting six hours) in a rarefied atmosphere and a physical load under these conditions. The latter was in all cases a standard load and consisted in the following: the examinee in a sitting position lifted in outstretched hands dumbbells weighing 4 kg apiece 20 times a minute, then climbed up on a 30-cm-stool 20 times a minute for 3 min and then lifted the dumbbells again for another minute. Thus, one cycle of the physical work occupied 5 min, and these cycles were repeated at 15-minute intervals throughout the staying time at the altitude. When staying six hours at the altitude, the examinee was occupied with performing physical work for 90 minutes. According to calculations, the energy value of the work amounted to about 250 kcal/h. /263

Inasmuch as the discussion has turned to the physical load employed in our investigations in a rarefied atmosphere, here it is clearly appropriate to dwell briefly on the general organization and methodology of our experiments. The investigations were carried out on official examinees, as well as on volunteers (mainly university students) -- men ranging in age from 23 to 30 years. All of the examinees appeared before a special medical examination board for permission to participate in pressure-laboratory investigations according to an approved program.

A greater part of the investigations were carried out in a GBK-63 pressure chamber (fitted out on the basis of an SBK-48 pressure chamber), a lesser part, in a TABAI-17 pressure chamber.

In each experiment, two examinees were placed in the first chamber; in the second, which was larger, from four to five examinees. Both chambers had air locks, permitted atmospheric rarefaction down to the necessary degrees and gaseous media with a changed oxygen and nitrogen content to be created (the GBK-63 chamber permitted us to work also with helium-oxygen media) and were equipped with regenerating devices ensuring carbon dioxide absorption and a system for automatically keeping the temperature and humidity at the required level. These latter parameters in our experiments were kept within the customary mean range (temperature 18-21°C, humidity 60-65%). Both chambers were equipped with apparatus for gas analysis and telephones for two-way communication between experimentor and examinees; the TABAI-17 pressure chamber also had two television apparatus so that the examinees could be observed not only through the porthole, but also from the operator's console.

The chambers were fitted out with cots for the examinees to rest on during long experiments and chairs for them to sit on during short experiments and in the final stages of the experiments. In the event that the examinees developed especially serious altitude decompression disorders that did not subside upon return to ordinary pressure, our main GBK-63 chamber was rigged up with an RMK-3 recompression chamber. Incidentally, the need to use it did not arise at any time during our experiments.

For 24 hours before the experiment, the examinees were placed in a hospital, where they were subjected to clinical examination by different specialists. In the hospital, they received food precluding the development of altitude meteorism. On the day of the experiment, after a light breakfast, the examinees were brought to the laboratory, and upon termination of the experiment, they were returned to the hospital, where they remained for another 24 hours.and underwent a final /264
clinical examination. At appointed times in the course of the long experiments, the examinees received food through the airlock (excluding, of course, the last periods of the experiments with a stay at altitudes of 7-11 km).

All experiments included two periods: the first, preliminary period, in the course of which the examinees were either in an ordinary atmosphere (breathing air or oxygen) or in one or another changed gaseous medium (in this connection, see below) and a second, main period, when the examinees, after switching on their oxygen equipment, were raised to an altitude (from 7 to 11 km, where they had to remain six hours (or four hours), performing the above-described physical work. Upon developing pronounced decompression disorders, which, as a rule, did not appear simultaneously in both (a fortiori, in the four or five) examinees, but developed first in one of them, the examinee in question was sent through the airlock, and descent was

begun -- the pressure was increased to its normal value. The possibility of alternate airlocking permitted a given experiment to be carried through to the end on all examinees participating in it. In all experiments, the respiratory and cardiac rate and the arterial pressure were recorded. In the first, preliminary stage of the experiment, if it was a long one, these physiological indices were recorded every hour, while in the final stage -- at the altitude -- they were recorded every 15 min. Besides this, in a number of experiments, a great number of special, in particular electrophysiological, indices were recorded. In the event that pronounced decompression symptoms appeared, additional recordings of the physiological indices were made immediately after the symptoms developed. Throughout the course of the examinees' stay at the altitude, a continuous watch was kept of them through the chamber porthole, and all outwardly noticeable changes in their condition, behavior and activity were recorded. The examinees' subjective condition was evaluated by recording their verbal reports and from the data in their written reports about their self-feeling while staying at the altitude and about the appearance of decompression symptoms of one sort or another, their nature, dynamics and time of disappearance incident to descent.

After these unavoidable methodological digressions, we must return to our main line of argument -- exposition of the factual material characterizing the development of altitude decompression disorders under typical space flight conditions.

It must be said that even before the advent of these conditions, facts were being accumulated that attested to the provocative influence of physical work on the development of altitude decompression disorders. Many such data were obtained by a great number of investigators [Armstrong, 1952; Henry, 1956; Bennett, 1961; Degner et al, 1965; etc.]. In experiments /265 by Kuznetsov, Poleschuk and coauthors on the effect of rarefaction corresponding to an altitude of 11 km without preliminary desaturation, the negative role of physical work became clearly apparent. In these experiments, decompression disorders developed in all of the examinees without exception in the first hour of staying at the altitude (most often on expiration of 15-20 min), and they were very pronounced. From these data and others gathered earlier it became indubitable that a low pressure equal to 170 mmHg (corresponding to an altitude of 11 km), which is the lower limit for a man's stay in a spacesuit, constitues a very serious threat from the standpoint of development of decompression disorders.

As is well known, different pressure values can be used in a spacesuit: from the above lower limit of 170 mmHg to 308 mmHg. Significantly, in choosing pressure conditions in the indicated range, a knotty dilemna always crops up: the lower the

pressure, the greater the mobility of the man in the spacesuit and consequently, the better the conditions for any useful work; but, at the same time, the greater the danger of decompression disorders. It was necessary to determine whether this danger exists in the case of the highest pressure used in spacesuits: 308 mmHg.

To elucidate this question, we carried out special investigations [Poleschuk, Kazakova, Katuntsev, Tokarev et al]. We conducted 107 experiments on 37 examinees with ascents to an altitude of 7 km (pressure of 308 mmHg) without preliminary desaturation. One program of 60 experiments was designed for a six-hour stay of the examinees at the altitude; another program consisting of the remaining 47 experiments, was designed for four-hour exposure. In all cases, we employed the above-described physical work. The experiments showed that under the indicated conditions, decompression disorders in the form of muscular and joint pains developed in more than half of the cases. They were detected in 58 of the 107 experiments. These 58 cases involve 29 examinees; the other 12 examinees had no pains. The latter circumstance attests to the role of individual differences in resistance to decompression effects; on this question, which is of such great practical importance, I shall dwell in greater detail below.

It should be noted that in conducting the series of experiments that we are now considering, as well as in other, similar series involving transition from normal pressure to an altitude and, likewise, as in our early investigations (self-experiments), it was customary to terminate the experiment only upon appearance in the examinee of rather severe, hard-to-bear painful sensations (or in the case of bad general self-feeling). When clearly perceptible, but weak and quite endurable, painful sensations developed, the experiment was continued until the symptoms acquired a pronounced form. With such an /266
organization of the experiments, it was possible to judge of the dynamics of the symptoms and to answer the practically important question of how events develop incident to the protracted action of rarefaction and how matters in this case stand with a man's working efficiency.

The experiments showed that altitude decompression pains seldom arise in an acute form right away. This confirms especially those relatively low altitudes that we are now talking about. As a rule, the matter does not even begin with pain, but with sensations of awkwardness or, one might say, some discomfort, in one or another region of an extremity -- most often, a region adjacent to some joint. These sensations gradually change into a clear feeling of pain, a pain that is at first evaluated as comparatively weak. It was symptoms of this kind -- the presence of clear painful sensations -- that we regarded in our experiments as signifying the development of decompression

disorders (the above-mentioned 58 cases out of 107). Subsequently, even at such a relatively low altitude as 7 km, the pain in most cases progressively intensifies and after some time becomes difficult to bear or even intolerable. Clear painful sensations that arose more seldom in one or another stage of the experiment do not intensify but even weaken in the course of time, and sometimes even vanish, in some cases without a trace, while in other cases, left behind is that sensation of awkwardness or discomfort with which the decompression disorder symptoms began. In such cases, the examinees were able to complete the program of the experiment, which was indeed accomplished, individual cycles of physical work sometimes being performed in certain periods of the experiment at the experimentor's behest. In 35 of the above-mentioned 58 cases of appearance of altitude pains, their progressive intensification to intolerable proportions was noted, the examinees could not complete the program of the experiment and the matter ended in their premature descent from the altitude. In 23 cases, the pains that arose were tolerable, did not intensify to extreme proportions and, in a number of cases, weakened or vanished with the passage of time, and the program of the experiments (in 16 cases, four-hour experiments and in seven cases, six-hour experiments) was completed.

In the following series of experiments, the effect of an altitude of 8,100 m was investigated. This altitude corresponds to a pressure of 263 mmHg, which amounts to about 0.35 atm.

We carried out 70 experiments on 39 examinees with ascents to the indicated altitude without preliminary desaturation involving in most cases a four-hour and, in some cases, a six-hour stay at the altitude and, of course, performance of physical work.

In only 26 out of 70 experiments were altitude decompression disorders absent (in 24 experiments with a four-hour exposure and two experiments with a six-hour exposure). In /267
44 cases involving 31 examinees, manifest decompression disorders developed in the form of very pronounced painful symptoms. In six experiments, immediately upon the appearance of these symptoms, the examinees were raised to a higher altitude (the results of this test will be discussed below). In 28 of the remaining 38 cases of decompression disorders, the painful symptoms intensified, became difficult to bear and necessitated immediate descent; but in ten cases, they did not progress or even weakened, and the program of the experiments was completed.

With regard to when the altitude decompression disorders (clear painful sensations) developed, all cases of them in the two just-mentioned series of experiments can be classified as follows:

Altitude, m	Time of stay at altitude					Total number of cases of decompression disorders
	1st h	2nd h	3rd h	4th h	5th h	
7,000	13	18	16	11	-	58
8,100	18	17	3	5	1	44

The above figures show that in the case of ascents to altitudes of 7,000 and 8,100 m without desaturation, decompression disorders can develop at very different, including widely-separated, times. If decompression symptoms develop at altitudes of 10,500-11,000 most often in the first 10-15 min after ascent and, in general, appear in the first hour of the stay at the altitude, then, at an altitude of 8,000 m, the maximum development of decompression disorders now covers the first two hours, while at an altitude of 7,000 m, it is shifted toward the second and third hours of the stay in a rarefied atmosphere. A considerable part of the cases of appearance of decompression disorders occur in the fourth hour of the stay at the altitude; we even encounter a case of symptoms developing in the fifth hour. Of theoretical interest and great practical importance is the circumstance that between the time of appearance of the painful symptoms and their intensity there is no constant dependence. Symptoms that appear at later times often progress rather rapidly and attain a critical intensity, whereas pains that developed early may undergo no development and may not lead to forced descent. However, if we evaluate the picture as a whole, in the case of painful decompression symptoms that develop early, there is always a greater chance that a forced descent will be required and that the program will not be completed.

The adduced facts show that when muscular work is performed under the prolonged (four- and six-hour) action of rarefaction, the entire range of reduced pressures that is envisaged for /268 spacesuits (from 170 mmHg -- 0.22 atm -- to 308 mmHg -- 0.4 atm) proves to be dangerous in respect of decompression disorders when a man changes into his spacesuit from normal pressure without any prophylactic measures. As regards the lowest pressures (230-170 mmHg, altitudes of 9-11 km), this danger was clear even before the requirements of space flights, if it was not even somewhat understated, because of the fact that in most previous investigations and in flying practice, no physical work took place under the indicated degrees of rarefaction. But up to the present time, the considerable and constant danger of decompression disturbances incident to lesser degress of rarefaction, in particular at a pressure of about 300 mm (altitude of 7,000 m), was unsuspected.

In the literature of the last two decades, reports were constantly being accumulated about individual cases of

decompression disorders at such altitudes as 7-6 and even 5 km [Kendricks and Gag, 1949; Mitchell and Edmond, 1951; Schneck, 1957; Odland, 1959; Fryer, 1964]. But most authors presented such cases as exceptions, and this was the very reason for their separate independent publication. This is precisely the way the above facts are interpreted in even the most recent studies (cf., for example, Bogoslovov, 1972). In other words, according to ideas that have prevailed up to the present time, the real danger of decompression disorders arises at altitudes in excess of 8 km. The adduced material shows that these ideas do not correspond to the actual state of affairs. If, for short-lived exposures at an altitude and relative muscular repose, these ideas can still be accepted with some reservations, for space flight conditions involving the prolonged action of rarefaction and physical work, they are patently unacceptable. The facts compel us to recognize that even for the highest pressure employed in spacesuits -- 308 mmHg (0.4 atm) --, the danger of decompression disorders under the just-mentioned conditions is rather great: they arise in more than half of the cases in the overwhelming majority of examinees.

It must not be forgotten that these figures refer to the concrete conditions of our experiments, in particular the physical work we chose; it is possible that for alternative forms of man's muscular work and working conditions in a rarefied atmosphere, the frequency of development of altitude decompression disorders will be somewhat different. But the probability of their development incident to a prolonged stay and physical work under conditions of rarefaction corresponding to altitudes of 8 km and even 7 km remains beyond question. It is important to stress that the development of the above-described decompression disorders is accompanied by considerable changes in man's working efficiency. Even in their first /269
stages involving relatively weak pains, an outside observer can clearly notice not only those manifest difficulties which the examinee experiences in performing routine muscular work and the imperfect execution of this work, but also such shifts in his general condition as attest to a reduced efficiency in the broad sense of the word (slowing down of reactions to different external stimuli and, in particular, verbal signals, inaccurate execution of commands given by the experimentor, a wandering of attention, etc). With intensification of the painful symptoms, all of these manifestations progress rapidly, and when the pains acquire an intolerable character, man's general condition and efficiency is very sharply impaired. On the basis of self-experiments, (at an altitude of 11, as well as 7, km), I can testify that when the stay in the rarefied atmosphere involving pronounced decompression disorders with painful symptoms is prolonged, the latter attain extreme proportions. Besides the fact that it becomes quite impossible to execute any number of movements in which the affected joint and muscles adjacent to it participate, the pain itself proves to be so

intense that it causes a great number of vegetative reactions (cold sweat, change in respiration and heartbeat) and paralyzes any purposeful, especially delicate, external activity.

Whence it follows that decompression disorders incident to an astronaut's working in a spacesuit in empty space or on the surface of heavenly bodies pose a very serious threat to the execution of a planned operation, as well as to the well-being of the astronaut himself.

Changes in the state of the central nervous system and upper nervous activity incident to development of altitude decompression disorders

The question of the changes in upper nervous activity and in the state of the central nervous system incident to development of decompression disorders is indubitably of great scientific and practical interest. There are at least three reasons for this interest. In the first place, we can take it for granted that the fine indices of the state and activity of the central nervous system can signal the development of decompression phenomena (the process of gas formation in the organism) even before pronounced decompression disturbances appear; in other words, we can assume that the above indices help to detect and bring out latent forms of decompression disturbances. In the second place, study of the indices of the state and activity of the nervous system before the onset of painful decompression symptoms, as well as incident to their development, may be promising from the standpoint of obtaining objective criteria for decompression disorders. And, finally, /270 in the third place, study of this question is undoubtedly important for characterizing the functional state of the central nervous system and the organism as a whole incident to the development of decompression phenomena and for evaluating man's efficiency under these conditions.

In spite of this considerable interest, the question of the state and activity of the central nervous system incident to decompression disorders has hitherto not been the subject of independent special study. In the literature, we find only separate studies in which, together with other physiological indices, the quality of the examinees' execution of psychophysiological tests was determined, while in other investigations, that electroencephalogram was recorded incident to compression and decompression effects [Roger, et al, 1955; Bennett, 1961, O'Conner and Pendergrass, 1966; Turygisa and Aleksandrov, 1968; Bennett and Towse, 1971; Fructus et al, 1971; Proctor, 1972]. It should be noted that none of the investigations connected with the increased pressure range were intended for analysis of decompression disorders. Ascertained in these studies is the overall effect

of compression and decompression factors. Strictly speaking, therefore, the obtained data are not devoid of contradictions and, on the whole, do not permit any definite conclusions to be drawn about changes in the electroencephalogram or shifts in the functional state of the central nervous system caused by decompression disorders. As far as studies of upper nervous activity and of the functional state of the central nervous system incident to development of altitude decompression disorders are concerned, none are to be found in the literature.

As has already been pointed out, in our early self-experiments involving study of decompression disorders incident to ascents to altitudes of 10.5-11 km without desaturation, a particular place was devoted to investigation of upper nervous activity. The general results concerning the development of decompression disorders in examinees that we obtained from these experiments were examined above, at the start of the present chapter. Now we must dwell on the results obtained incident to studying upper nervous activity.

The latter was studied in four examinees by the method of motor conditioned reflexes to verbal reinforcement. The motor reflexes consisted in the examinees pressing the button of an electric switch. As conditioned stimuli sound and light signals were used. The sound stimuli were produced through headphones with the aid of a special sound generator; the light signals, with the aid of colorless and colored 26-volt electric bulbs placed on a board before the examinees. Effective, trace and discriminatory conditioned reflexes were investigated. Incident to investigation of effective reflexes, the examinee, according to the instruction, had to press the switch immediately upon presentation of the light or sound signal; incident to investigation of trace reflexes, two seconds after presentation of the light signal. Incident to investigation of discriminatory reflexes, red, blue and yellow lights were used. One of them, according to the instructions, served as inhibiting stimulus, and the others, as positive stimuli. The presentation of the stimuli and responses of the examinees were recorded with the aid of an apparatus built in the pressure laboratory of the VMOLA [S. M. Kirov Military Medical "Order of Lenin" Academy] by U. L. Komendantov and N. N. Sekilstov. On the rapidly moving waxed tape of this apparatus, one electromagnetic marker recorded the time every 0.05 sec (50 sigmas), while others recorded the presentation of the different kinds of signals and the responses of the examinee. Reflex time could be taken into account with an accuracy of up to 25 sigmas inasmuch as the segment between two neighboring time marks on the tape could easily be divided in two.

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Incident to each determination of the indices of upper nervous activity, first effective, then trace and finally discriminatory

conditioned reflexes were investigated. Incident to investigation of effective reflexes, the light and sound stimuli were presented alternately at 1.5-sec intervals. On each occasion, 10-12 signals of each kind were used. In connection with the fact that the stimuli acted in a definite rhythm, a stereotypy of reflexes rhythmically succeeding one another could be formed in the examinees. To check the examinees' ability to suppress this stereotypy, individual stimuli were presented either somewhat earlier or somewhat later than the usual half-second time. Incident to investigation of trace reflexes, the trace stimulus (colorless bulb) also acted 10-12 times at intervals of from 3 to 6 sec. In investigating discriminatory reflexes, 8-10 positive stimuli were presented and alternately with them, 3-4 inhibiting stimuli. The intervals between stimuli varied from 3 to 5 sec. To avoid the formation of stereotypy in the examinees, the order of presenting these stimuli was changed from time to time.

The investigation of reflexes in each separate case occupied about 6 min. It was conducted four times in each experiment: immediately before ascent, after staying 10 and 30 min at the altitude and 10 min after termination of descent.

Fig. 72 shows sample recordings obtained in investigating conditioned reflexes. Recorded in each separate case as a result of the investigations were the mean time of the effective reflexes to sound, and, separately, to light, the mean time of abandonment of the trace reflex and the number of incorrect responses on the part of the examinees.

In all cases without exception of pronounced decompression disorder symptoms in the form of itching and muscular and joint pains, considerable changes were noted in the investigated indices. In the first place, as a rule, the mean value (in sigmas) of the latent periods of the motor conditioned reflexes to the light and sound stimuli lengthened considerably, as is clearly indicated by the following data: /272

	On the ground		At an altitude of 10,500-11,000 m	
Examinee	To light	To sound	To light	To sound
1	253	231	318	309
2	244	228	264	284
3	223	190	243	186
4	281	259	311	282

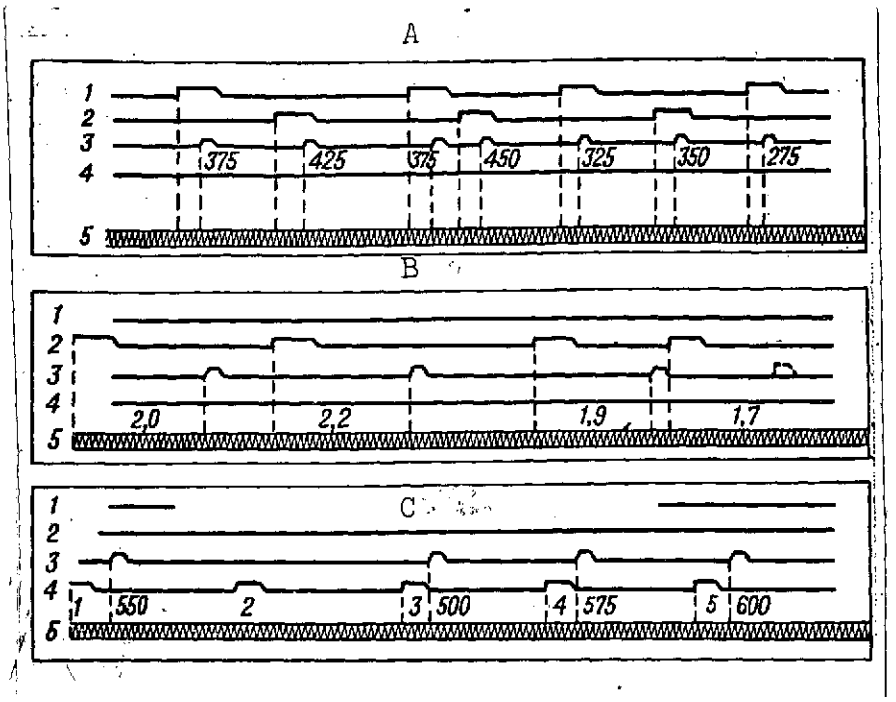

Fig. 72. Sample recording of motor conditioned reflexes on Komendantov-Feklistov instrument. A -- recording of effective reflexes; B -- recording of trace reflexes; C -- recording of discriminatory reflexes; 1 -- reflexes to sound; 2 -- reflexes to light; 3 -- responses of examinees; 4 -- presentation of discriminatory stimuli; 5 -- time mark, 50 sigmas.

Incident to development of decompression symptoms in the form of itching and pains, in all four examinees the latent period of reflexes to light, and in three, the latent period of reflexes to sound, turned out to be increased by tens of sigmas. The only exception is the mean latent period of /273
sound reflexes in the presence of decompression phenomena in examinee No. 3 -- it is almost equal to its initial value. As can be seen from the table, especially considerable is the increase in the latent period of effective reflexes in examinees Nos. 1 and 2. And the decompression disorders generally took a considerably more serious course in them than in examinees Nos. 3 and 4.

In the second place, incident to the development of decompression disorders, the relations between the latent periods of reflexes to sound and light stimuli changed in a characteristic manner. As a rule, incident to the determination of the initial background, the time of responses to sound stimuli proved to be less than to light stimuli. Incident to development

of pronounced decompression disorders, this difference usually decreased or leveled out, while in a number of experiments the temporal relations became reversed. This is confirmed by the figures of Table 15.

TABLE 15. CHANGE IN RELATIONS BETWEEN LATENT PERIODS OF REFLEXES TO SOUND AND LIGHT STIMULI INCIDENT TO DEVELOPMENT OF DECOMPRESSION SYMPTOMS IN EXAMINEES AT ALTITUDES OF 10,500-11,000 m.

Examinee	Experiment number	Value of latent periods in % of initial level: After staying 30 min at the altitude (incident to development of decompression symptoms) Sound	Light	10 min after descent Sound	Light
1	6	185	150	116	93
1	12	142	117	83	115
2	12	131	111	95	102
2	18	117	100	100	105
4	8	108	86	71	93
4	20	135	115	112	95

In these experiments, the temporal relations between the values of the latent periods of sound and light reflexes as compared with their initial values became reversed: the latent periods of reflexes to sound turn out to be greater than those of reflexes to light. Analysis of the dynamics of these shifts in individual experiments shows that as decompression disorder symptoms develop and intensify, the latent periods of reflexes to light and sound first level out and then, incident to acute painful sensations, the temporal relations between these periods become reversed. It should be noted that in examinee No. 3, in whom decompression disorders took a milder course than in all the rest, no such shifts in the latent periods of reflexes were detected.

The third change that is characteristic of pronounced cases of decompression disorders is an increase in the fluctuations in the latent periods of individual effective /274 reflexes in the course of each determination. If, before ascent, the maximum difference between the shortest and the longest response to the one stimulus or the other usually amounted to about 100-150 sigmas, then, incident to development of decompression symptoms, it reached 400-600 sigmas and more.

Fig. 73 shows the results of one of the experiments (No.6), in which intensive itching and severe pains developed in examinee No. 2 at the altitude. The curves show the distribution of the number of reflexes versus their latent period before ascent (solid line), at the altitude in the presence of caisson pains (broken line) and after descent (dot-and-dash line). Conspicuous is the wide range of fluctuations in the latent periods of individual reflexes during decompression pains at the altitude (broken lines).

The fourth regular phenomenon characterizing upper nervous activity incident to decompression disorders is an increase in the number of errors by examinees incident to investigation of conditioned reflexes. The errors were recorded when using positive and discriminatory colored light signals (impaired discrimination), as well as incident to omission of recurrent stimuli of the effective reflex and when using extrastimuli (earlier than the time of recurrence). In the last two cases, the errors took the form either of responses without the action of a stimulus (reflex to rhythm) or, in their absence, incident to the action of the extrastimuli. Data on the errors made by examinees incident to development of pronounced decompression disorders are shown in Table 16.

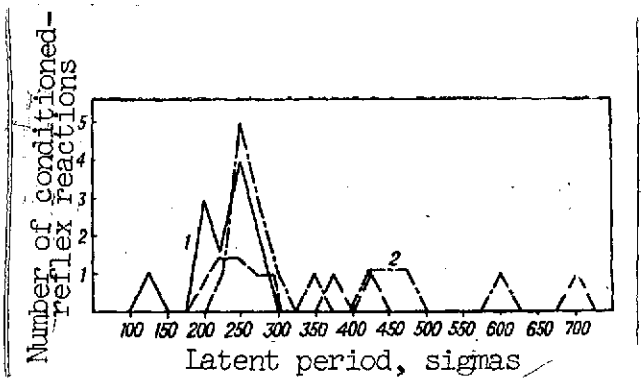

Fig. 73. Distribution of number of motor conditioned reflex reactions versus duration of latent period. 1 -- control; 2 -- experiment.

As a rule, incident to development of prolonged decompression disturbance symptoms, the mean time of the trace reflex changed. These changes were twofold. In some cases (most often, incident to especially severe pains), the mean time of the trace reflex was shortened; in other cases, it was lengthened (most often incident to pains of lesser intensity). Evidence that the noted changes in upper nervous activity are mainly connected with decompression disorders, and not with hypoxemia, is the fact that these changes are absent from examinees who felt no signs of decompression disturbances or experienced only weak itching at the altitude. This is also borne out by the results of the control experiments, in which examinees were raised to an altitude of 5,000-5,500 m without oxygen. There can be no doubt that in this case, hypoxemia attained higher intensities than at altitudes of 10,500-11,000 m incident to oxygen breathing, while the changes in upper nervous activity in the experiments at 5,000-5,500 m

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TABLE 16. NUMBER OF WRONG REFLEX REACTIONS IN FOUR EXAMINEES INCIDENT TO DEVELOPMENT OF DECOMPRESSION DISTURBANCES AT ALTITUDES OF 10,500-11,000 m.

Examinee	Before ascent			At the altitude incident to muscular and joint pains		
	Impaired discrimination	Responses to rhythm	No reaction	Impaired discrimination	Responses to rhythm	No reaction
1	1	4	1	3	11	3
2	—	—	—	1	1	7
3	2	2	—	3	7	—
4	4	1	2	8	4	4
	7	7	3	15	23	14

were generally not so significant as incident to acute decompression disorders, and were sometimes notable for their character. Incident to hypoxemia at intermediate altitudes, the latent period of effective reflexes generally changed in two phases: at first it was shortened, and then lengthened; but incident to decompression phenomena at higher altitudes, the shortening of effective reflex time took place only in individual cases.

In the overwhelming majority of cases, altitude meteorism was manifested only in some increase in volume of the stomach and was not accompanied by painful sensations. But swelling of the stomach occurred not only in those experiments in which the examinees developed decompression disturbances and the above-described changes in conditioned reflexes, but also in other experiments, when either the one or the other was noted. Consequently, altitude meteorism as well as hypoxemia, could not be the cause of the above-mentioned shifts in conditioned-reflex activity.

In terminating our account of the experimental data concerning upper nervous activity, we must dwell on still another fact that, in our opinion, is of definite significance. Noted in some experiments in examinees still on the ground, incident to deter-/276 mination of the initial state of conditioned-reflex activity, was a smoothening out and reversal of the temporal relations of the latent period of the effective reflex to sound and light stimuli. Incident to ascents to altitudes of 10,500-11,000 m,

in these cases decompression disorders arose in the examinees more often, developed more rapidly and were manifested with greater intensity.

In addition to the foregoing, it should be noted that in the course of the investigation, clear changes in the resistance of individual examinees to decompression phenomena were detected. In this case, the resistance of different examinees changed in different directions: in examinees Nos. 1 and 2, it decreased in the course of the investigation, while in examinee No. 3, it increased appreciably. These changes in resistance were in complete agreement with how the examinees subjectively evaluated their general condition at the corresponding periods of the investigation. Fatigue and deterioration of self-feeling corresponded to their low tolerance of the experimental conditions and vice versa. Significantly, simultaneously with this, the indices of upper nervous activity that we investigated also changed incident to their determination under ordinary conditions before ascent. At periods when the examinee noted fatigue and when his resistance to decompression effects decreased, even before the start of ascents considerable fluctuations in effective reflex time and a smoothening out and reversal of the temporal relations of the reflexes to light and sound were detected.

Thus, the results of these experiments involving study of upper nervous activity permit us to conclude that the latter undergoes considerable changes in the course of development of decompression disorders and show the need for in-depth study of the state of the nervous system under the conditions in question.

To this end, last year special investigations were begun in our team by L. V. Kalyuzhnyy and his collaborators -- A. A. Antonov, G. P. Goroyan, I. N. Vakharova and N. M. Kirgizova. In these investigations, which were conducted on people, the electroencephalogram of the frontal, parietal and occipital regions of the cerebral cortex, the potential elicited by a flash of light and the monosynaptic, so-called H reflex obtained by electrostimulation of the tibial nerve were studied. The investigations were carried out V. P. Nekrasov and his coworkers, who evaluated the state of the central nervous system of examinees and their efficiency by a number of psychophysiological tests (work with Schultz-Platonov tables, solution of arithmetic problems, memorizing, etc). In these experiments, ascents were effected to an altitude of 11,000 m without desaturation, with the examinees breathing through a mask from the beginning of ascent. The experiments were conducted in the following manner. At the start of the experiment, the initial values of the above-mentioned indices were recorded under ordinary pressure, then the examinees switched on their oxygen equipment and were raised /277

to an altitude of 11,000 m at a rate of 15-20 m/sec; at the altitude, all of the indices were recorded in the initial period, and then incident to the onset of painful decompression symptoms, which usually appeared after muscular work.

This necessitated keeping the examinee at the altitude for some time while he was feeling pains -- of course, not unduly severe, but endurable. After this, he was lowered to an altitude of 2-3 km. The pains disappeared in the process of descent, and at an altitude of 2-3 km, all of the indices were recorded. Then ascent was repeated, in the course of which, usually at altitudes of 7-9 km), painful symptoms reappeared. When they set in , the ascent was interrupted and the indices were recorded once again, after which the examinee was lowered to the ground (the pains again disappeared in practically all cases) and the data were recorded for the last time. In all, 79 experiments were conducted on 18 examinees. Painful decompression symptoms arose in almost all experiments and were mainly localized in the lower extremities, most often in the knee joints.

The changes in the background electroencephalogram in the course of the experiments were ambiguous in different examinees. The examinees were divided into two equal groups (nine men in each) on the basis of the character of the initial background electroencephalogram. The first group consisted of examinees whose electroencephalogram was characterized by a pronounced alpha rhythm with open, as well as closed, eyes.

The alpha rhythm in examinees of this group amounted on the average to 28 $\pm$ 1.5%, and the theta rhythm, to 24 $\pm$ 1.5%. In the second group of examinees, the theta rhythm dominated (30 $\pm$ 0.74%), while the alpha rhythm was present to a lesser degree (21 $\pm$ 0.75%). Incident to the first recording at an altitude of 11,000 m, even before the onset of decompression symptoms, changes in the electroencephalogram were detected in more than half of the cases (in 57%). For the most part (33%), they were manifested in an increase in the percent content of the delta and theta rhythms and a decrease in alpha activity. In a smaller number of cases (27%), on the other hand, they were manifested in an increase in alpha activity. These changes in the electroencephalogram, opposite though they are, deserve attention inasmuch as they belong to the period preceding the development of decompression disorder symptoms.

Their further experimental analysis ought to distinguish the possible influence (besides very probable incipient decompression phenomena) of other altitude factors, such as expansion of gases in the intestine and some hypoxemia, which takes place at an altitude of 11,000 m in spite of breathing pure oxygen. As far as decompression disorders are concerned, here it is

impossible to exclude general aeroembolic phenomena inasmuch as the experiments were conducted without preliminary desatura- /278
tion. Incident to the appearance of painful decompression sensations in the examinees, the changes in the electroencephalogram were more pronounced and definite. In 73% of the examinees of the first group (with a predominance of the alpha rhythm in the initial recordings), alpha activity decreased and a predominance of the delta and theta rhythms was obtained on the electroencephalogram. In the examinees of the second group, the delta rhythm also increased, but at the same time, the theta and alpha rhythms decreased. The recorded changes in the frequency composition of the electroencephalogram was statistically significant. Thus, most typical of painful decompression symptoms was a shift in the frequency spectrum of the electroencephalogram to the left, toward lower frequencies. After descent to altitudes of 2-3 km, simultaneously with the disappearance of painful sensations the electroencephalogram also became normalized in the greater part of the cases (61%), but at this stage of the experiment, the increase in delta and theta waves was maintained in 39% of the cases. Incident to reascent, pains reappeared at altitudes of 6-7-8 km and so a halt was effected at these altitudes and the electroencephalogram and other indices were recorded. In so doing, in 82% of the cases changes were noted in the electroencephalogram which were analogous to those which arose incident to the initial pains, and not seldom even more pronounced. This fact permits the indicated changes in the electroencephalogram to be definitely connected with decompression pains, and not with any other effects of rarefaction.

Immediately after descent to the ground (painful sensations disappeared completely in the very process of descent), in 77% of the cases the electroencephalogram did not differ in character from the initial one recorded at the start of the experiment. In 23% of the cases, in the first recordings after descent, changes in it were still noted that were similar to those at the altitude; these changes smoothened out and disappeared 30-40 min after descent. Noteworthy are data on changes in the electroencephalogram when performing psychophysiological tests requiring mental effort. When performing such tests, the electroencephalogram of most examinees showed some predominance of delta and theta rhythms. At the altitude, this phenomenon proved to be more pronounced than under terrestrial conditions.

The potential elicited by a light stimulus showed appreciable changes in the period before the appearance of painful decompression symptoms and, especially, incident to development of these symptoms. These changes concerned the latent periods of different components of the elicited potential and of the energy of their wave components. At an altitude of 11 km, even before the appearance of pains, in 70% of the cases a shortening

of the latent periods of all six waves of the elicited potential was noted, this shortening being statistically significant for waves III and IV. Incident to the appearance of pains, the /279 latent periods of all waves of the elicited potential were shortened even more, and this shortening was statistically significant for all waves except the second. In the remaining 30% of the cases, before the appearance of pains the latent period of some waves (I, II and III) of the elicited potential lengthened appreciably, while others (IV, V, VI) shortened somewhat. But incident to the onset of pain, the latent periods of all waves of the elicited potential turned out to be lengthened as compared with the background.

Significantly, in two of the cases under consideration the initial values of the latent periods of the waves of the elicited potential were different. In the first case, in which a shortening of the latent periods of the waves of the elicited potential took place incident to decompression pains, their initial values were greater, and, conversely, in the second case, in which the pains were accompanied by a lengthening of the latent periods, their initial values were less. In other words, short latent periods incident to development of decompression disorders lengthened, while long ones shortened, these changes beginning, as has already been said, even before the appearance of pains and intensifying incident to pains.

The authors judged of the energy of the waves of the elicited potential by continuing the wave to its height. This index also began to change even before the appearance of pains, but in this period, the shifts for individual waves were different. Incident to the onset of pains, a reduction in energy (by 10-30%) was typical of all waves except the sixth, whose energy increased in a number of cases by more than 100%.

It should be noted that the above-described shifts in the character of the elicited potential are the most typical. In individual experiments, different combinations of changes in the components of the elicited potential are encountered, and there are individual differences in the reaction. I shall not enter into the details inasmuch as the material is in the stage of analysis and processing. Detailed study of the detected changes in the electroencephalogram and of different components of the elicited potential is interesting and promising from the standpoint of fine analysis of the interrelations accumulating between different divisions of the central nervous system in the course of development of decompression disorders.

Ascertainment of the intimate mechanisms and internal genesis of the detected shifts in the electroencephalographic indices will make it possible to give a more in-depth characterization of the functional state of the central nervous system and of man's efficiency incident to decompression

disorders and may shed light on the general pathogenesis of their development. This is a task for the near future. For the time being, however, it should be noted that the above-adduced fundamental results of studying the electroencephalographic indices is important in that they concern not only that period /280 of decompression disorders when pains arise, but also the foregoing symptomless period.

Also significant is the fact that between the above-adduced changes in electrophysiological indices and the results of using psychophysiological tests a correlation can be seen which emerges most clearly in the early stages of the experiment. Thus, at an altitude of 11 km, even before the appearance of painful symptoms, together with the above-mentioned changes in the electroencephalographic indices a deterioration of attention, memory and thinking processes (lengthening of the time to solve problems) was noted. Incident to the appearance of pains, these shifts were very pronounced; incident to the subsequent descent to an altitude of 2-3 km, they smoothened out somewhat, but did not disappear. It was obvious that, together with these changes in the brain's upper functions, which are due to the development of decompression disorders, in the course of the experiment the examinees developed fatigue with the passage of time. This can apparently be explained by the fact that psychophysiological tests in the final stage of the experiment, when the examinees had returned to normal pressure and all decompression disorder symptoms had been completely liquidated, generally yielded somewhat worse results than the initial ones obtained before the experiment.

The above data can be supplemented by the results of studying the monosynaptic H reflex. According to the data of Hoffman [1918], who originated the given method, as well as the data of other investigators who used it [Magladery, 1951; Vurfinkel' et al, 1965; Kots, 1972], the H reflex is a very sensitive test for evaluation of the activity of the reflex apparatus of the spinal column and of the function of the effector neuromuscular apparatus. As is well-known, in studying the H reflex, two fundamental indices are taken into account and compared: the initial contraction of the muscles that is due to impulses travelling along the efferent fibers of the stimulated nerve toward these muscles -- the so-called M response -- and their repeated contraction, which is caused by the passage of the impulse through the spinal reflex arc -- the so-called H response.

In the experiments under discussion, the H reflex was caused by electrostimulation of the posterior tibial nerve with rectangular pulses lasting 1 msec, the responses of the gastrocnemius muscle being recorded on a VC-7 oscillograph. The H reflex was recorded in the experiments under consideration before ascent to an altitude, and then at the altitude incident to the

appearance of pains and, further, in the process of descent, right down to complete disappearance of the painful sensations, which generally took place at altitudes of 2-3 km. The results turned out to differ as a function of whether the painful sensations developed in that extremity in which the H reflex was recorded or in other regions--in particular in the contralateral extremity. When the reflex was recorded in the extremity /281 in which the decompression pains arose, in 80% of the cases a clear and statistically significant decrease in amplitude of the H response by 17-29% of its initial value was detected. But when the reflex was recorded in the extremity in which the pain did not arise, incident to development of painful symptoms located elsewhere (in particular, in the contralateral extremity), in 80% of the cases the H response increased on the average by 12%; in 20% of the cases, a decrease in its amplitude was noted. In no case did the value of the direct response (M response) change.

The adduced facts show that in no case, including that in which the decompression pains are localized in the extremity in which the reflex is studied, does their development lead to changes in the functional state of the effector part of the reflex arc. Impulses directed straight at the muscle yield the same effects as under normal conditions (the M response remains unchanged). But the reflex responses prove to be clearly decreased incident to development of pains in the examined extremity in 80% of the cases; incident to pains located elsewhere, on the other hand, in only 20% of the cases. The matter therefore reduces to changes in the spinal reflex itself, which are apparently connected with its central closing link: transmission of impulses from the sensory to the motor neurons of the spinal cord. The fact that the changes in the reflex responses are noted in some cases when the pains are localized beyond the examined extremity compels the assumption that the mechanism of these changes is of a reflex nature. For the time being, only hypotheses can be made as to whether the corresponding reflex effects caused by painful decompression stimuli are confined to spinal reflexes, or whether they have a more complex path and descend from the brain or whether they arise as the result of a combination of both effects. At the same time, just as in the case of analysis of the electroencephalographic indices, we must leave out of account the possibility of local decompression phenomena (tissue or vascular gas formation) in these divisions of the brain. The results of studying the H reflex are significant from the practical standpoint because they disclose still another of the elements that determine changes in man's efficiency incident to decompression disorders. There can be no doubt that shifts in the functioning of the spinal reflex apparatus make a definite contribution to these general changes in man's efficiency.

Summing up all that has been said in the present section, we can conclude that the development of altitude decompression

disorders is accompanied by considerable changes in conditioned-reflex activity, the upper functions of man's brain and the functional state of different divisions of the nervous system. All of these changes are important, if not determinative, components of the general shifts in man's efficiency incident to /282
development of decompression disorders. They can shed light on questions that are very important from the practical point of view: how do the functional resources of the human organism change under the effect of decompression disorders, and to what extent will the working efficiency of an astronaut be curtailed and limited incident to development of decompression phenomena? Of particular interest in this connection, as has already been repeatedly stressed, is the period that precedes pronounced decompression symptoms. The facts show that in this period functional shifts in the upper divisions of the brain already take place and, consequently, changes in efficiency already set in.

In experiments conducted without desaturation, this can presumably be explained by general aeroembolic phenomena, which sets us the task of studying this question in investigations with desaturation and using such conditions of transition to the low pressures used in spacesuits as practically preclude aeroembolism and radically remove or prevent the development of painful symptoms. Insofar as latent forms of decompression disorders exist and no direct experimental data are yet at hand to reject the hypothesis of gradual growth of gas seeds from the very beginning of decompression, there still remains the question as to whether man's upper functions and efficiency will not change under low pressure conditions even in the absence of any clear and typical signs of decompression disturbances. This question is interesting from the theoretical, as well as the practical, point of view. To the foregoing we must add that the obtained data are encouraging from the standpoint of working out objective criteria for the onset of initial, as well as pronounced, decompression disorders, which is very important for their further experimental study.

Thus, this course of treating the problem of decompression disorders, which so far has undergone no independent development anywhere, is rather promising.

Experimental checking of the efficiency of desaturation for decompression disorder prophylaxis as applied to space flight conditions

It follows from the data of aviation physiology and flying practice that desaturation of the organism from nitrogen by breathing oxygen is a rather efficient means of altitude decompression disorder prophylaxis incident to a man's relatively

short-lived stay in a rarefied atmosphere without a physical load.

As has already been pointed out more than once, decompression/283 disorder prophylaxis on space flights involving the emergence of suited astronauts into empty space must be envisaged for different conditions, namely: the protracted effect of low pressures while a man performs muscular work.

The necessity of performing muscular work under conditions of rarefaction is undoubtedly a very serious circumstance that complicates the task of decompression disorder prophylaxis. As has already been said, the effect of muscular work, which provokes altitude decompression disorders, already put in an appearance when researchers first became familiar with altitude pains; in particular, in the above-mentioned self-experiments of Barcroft and his colleagues [1931].

In 1944-1945, Whitaker and his coauthors and, chiefly, Harvey and his collaborators laid the groundwork for experimental and theoretical study of the provocative effect of muscular work on the development of altitude decompression disorders [Harvey et al, 1944; Whitaker et al 1945]. Whitaker and his coauthors in experiments on frogs, and Harvey and his collaborators in experiments on cats, showed that muscular contractions to a great extent facilitate the formation of gas bubbles in the blood of animals incident to the effect of a rarefied atmosphere. This fact was explained by two circumstances: the intensified production of carbon dioxide incident to muscular work and its participation in bubble formation and the appearance as a result of muscular contractions of "gas seeds" predetermining bubble formation. The fact of intensified gas-bubble formation in a rarefied atmosphere incident to muscular contractions provided Harvey with one of his principal arguments in favor of the conception he developed of the significance of "gas seeds" in postdecompression gas formation.

Subsequently, incontestible experimental data began to accumulate which attested to the fact that altitude decompression disorders in people incident to a physical load arise considerably more frequently and take a more serious course [Fulton, 1951; Boothby, 1952; Henry , 1956, Haymaker, 1957; Hartman and Wunsche, 1961; Jones, 1967; etc]. It became clear that incident to a physical load, short-lived desaturation does not prevent the development of altitude decompression disorders. Behnke asserted that four-hour desaturation completely protected man from decompression disorders even incident to intense physical work at an altitude for four hours. This assertion, as we shall see, did not bear experimental checking. Nevertheless, even in a comparatively recent synoptic study devoted to decompression disorders [Behnke, 1971], the author rather optimistically speaks out in

favor of the efficiency of oxygen breathing as a means of preventing altitude decompression disorders and does not even raise the question of the acceptability of this means under space flight conditions. /284

At the same time, different opinions are encountered in the literature. Thus, Jones [1951] found that after two-hour desaturation the stay of examinees at an altitude of 11,500 m at rest for 90 min led to development of decompression disorders in 1% of the cases; but when physical work was performed at the altitude (stepping up on a 23-cm-high footboard nine times every five min), they developed in 40-50% of the cases. On the basis of these facts, the author made a very unfavorable prognosis for the efficiency of desaturation under the condition of a physical load at an altitude. He assumed that under this condition, not only four- but even seven-hour desaturation will not completely prevent altitude decompression disorders from developing. But the author did not check his assumption experimentally.

Individual cases of the appearance of altitude symptoms of the bends under a pressure of about 180 mmHg after preliminary desaturation lasting on the whole six hours were published by Beard and his coauthors [1967]. Their experimental plan was predicated on special applied tasks and provided for the examinees to breathe oxygen for four hours under ordinary pressure, then for two hours under a pressure of 258 mmHg, after which the examinees were subjected to a pressure of 180 mmHg for 15 minutes, during which time they performed five deep squattings and five pushups every five minutes. In three cases out of 70, decompression pains were noted. These data were very alerting in this respect that decompression pains, albeit in a small number of cases, developed after prolonged desaturation in the course of a very short (15-min) stay under a pressure of 180 mmHg. But as far as the total desaturation time of six hours is concerned, it must not be equated with the same desaturation time on the ground. The pressure of 258 mmHg, under which desaturation was effected for the last two hours, is dangerous in respect of decompression disorders. It is very probable that under this pressure, in spite of the preceding four hours of desaturation on the ground, the examinees developed gas bubbles which, under these conditions, did not yet manifest themselves, but displayed their effect incident to additional rarefaction down to 180 mmHg.

Thus, by the end of the Sixties a number of facts and opinions had accumulated in the literature which raised doubts about how acceptable desaturation of the organism from nitrogen by means of oxygen breathing would be for decompression disorder prophylaxis under space flight conditions. But this question, which had acquired a great practical acuity, required

for its elucidation special systematic investigations. Such investigations were carried out by A. K. Kuznetsov, I. P. Poleschuk and their collaborators in 1967-1969. /285

The authors studied the development of decompression disorders in examinees raised to an altitide of 11 km (a pressure of 170 mmHg) after different desaturation times within the framework of that experimental program which we have already talked about and which was adhered to in all of our subsequent investigations (six-hour exposure involving performance of the above-described muscular work) (Table 17).

TABLE 17. ONSET OF DECOMPRESSION DISORDERS AT AN ALTITUDE OF 11 km USING A PHYSICAL LOAD WITH DIFFERENT PRELIMINARY DESATURATION TIMES (ACCORDING TO DATA OF A. G. KUZNETSOV, I. P. POLESCHUZ, ET AL)

Duration of desaturation, hours	Number of experiments	Number of examinees	Number of altitude disorders (pains)	Time of appearance of altitude decompression disorders
2	15	13	15	2 min - 56 min
2	5	5	5	1 h - 2 h 20 min
3	3	3	3	2 h - 3 h 35 min
4	10	9	9	46 min - 3 h 41 min
5	6	6	4	20 min - 4 h 53 min
6	14	11	8	1 h 17 min - 4 h 05 min
7 and 8	4	2	2	1 h 13 min - 1 h 24 min

The adduced data show that incident to two-, three- and four-hour desaturation, decompression disorders arose practically in all cases (one experiment with four-hour desaturation was an exception). Thus, two-, three- and four-hour desaturation is not effective in preventing decompression disorders. It only postpones the time of their onset, which is borne out by the adduced extreme times of appearance of painful symptoms in different versions of the experiments. As far as the intensity of the painful sensations is concerned, desaturation on the whole does not yield any clear, positive, pain-relieving effect, either. Very often the painful symptoms that arose at late times rapidly increased to pronounced proportions. In none of the examined cases was the experimental program completed.

Incident to five- and six-hour desaturation, the frequency of onset of decompression disorders decreased somewhat, but they appeared in more than half of the cases; incident to seven- and eight-hour desaturation, they arose in two cases out of four.

Thus, the experiments of A. G. Kuznetsov, I. P. Poleschuk and coauthors, which were carried out in 1968-1969, clearly showed the insufficient effectiveness of desaturation, even when it lasts a long time, for altitude decompression disorder prophylaxis incident to the prolonged stay of examinees at an altitude while performing physical work. /286

At the same time, new and unexpected facts emerged, indicating that altitude decompression disorders after many hours of desaturation may develop after a very long staying time at an altitude -- in the fourth and even fifth hour of action of the rarefied atmosphere. Inasmuch as the examinees continued to breathe oxygen at the altitude, and, consequently, desaturation also continued, its general times under these conditions turned out to be equal to 12-13 hours. If we take the organism as a whole, with such desaturation times its desaturation from nitrogen must be so considerable that it retains only an extremely small part of the nitrogen contained in it. All these facts showed that altitude decompression disorders are characterized by very interesting distinctive features that demand careful attention, analysis and comparison with all the data accumulated earlier concerning that degree of desaturation of the organism as a whole and of its tissues at which the danger of decompression disturbances recedes.

When I and the team of collaborators participating in the collection of the above-mentioned facts were set the task of continuing our investigations, our first thought was to employ some additional means to increase the efficiency of desaturation. Having undertaken more experiments with six-, seven- and eight-hour desaturation and having convinced ourselves that they yielded the same results as before, we decided to go over to various modifications of the experiments. The first thing that we had to check was the possible significance of nitrogen diffusion through the cutaneous integuments of the examinees. As was mentioned earlier, in the experiment of A. P. Brestkin and A. G. Zhironkin [1959] it was shown that in those cases where the examinee breathes pure oxygen for a long time while his body is surrounded by an air medium, a constant flow of nitrogen from the surrounding atmosphere enters the organism through the cutaneous integuments and, further, through the blood and lungs. The quantity of nitrogen passing through man's organism under such conditions amounts, according to the data of the authors, to about 100 ml/h.

In connection with these data, it was natural for the thought to arise that, perhaps, this phenomenon of nitrogen penetration through the skin into the organism restricts the completeness of its desaturation from the given gas. Elucidation of this question was entrusted to R. T. Kazakova. Under the immediate guidance of I. P. Poleschuk and with the

participation of S. I. Tokarev, N. Yu. Leont'yeva and other collaborators, she undertook experiments in which, while examinees breathed pure oxygen during desaturation on the ground, as well as during their subsequent stay at an altitude, their body was also surrounded by an oxygen medium. To this end, a special light airtight suit was prepared, which was put on the examinee /287 and filled with pure oxygen at a low overpressure and a constant ventilation (5-6 ℓ min). Desaturation under these conditions was effected for 5, 6 and 8 hours, after which the examinees were raised to an altitude of 11 km within the framework of the usual experimental program to which we have alluded earlier (Table 18). Fifteen of these experiments were carried out on eight examinees, and in 11 of them, development of decompression disorders was recorded.

If these data are compared with the results of the experiments with desaturation under ordinary conditions, it will be obvious that there are no essential differences between them. Thus, the experiments in which a man's body was surrounded with oxygen showed that nitrogen penetration into the organism and the presence of an air atmosphere around the man's body during desaturation exerts no appreciable effect on decompression disorder development in the final stage of the experiment. Clearly, the nitrogen penetrating through the skin does not reach those tissues and regions in which are formed the gas bubbles that cause development of painful decompression symptoms. It is probable that the nitrogen diffusing into the organism from without takes a relatively short and direct path, getting mainly into the subcutaneous vessels and passing with the blood flow into the lungs. Clearly, this type of nitrogen circulation ought not to have a telling effect on the advent of altitude decompression disorders.

The next attempt to increase the effectiveness of desaturation was to combine it with physical work. In the course of six hours of desaturation, the examinees performed the same physical work that was used in all of the experiments (in particular, the present ones) during their stay at the altitude. Five such experiments were conducted on four examinees, and in all of them, decompression disorder development was recorded. In three experiments, six-hour desaturation while performing muscular work was effected at an altitude of 5,000 m; two of these experiments also terminated in decompression disorder development incident to ascent to an altitude of 11,000 m. One of these experiments deserves special attention because in it, the examinee, while still at an altitude of 5,000 m (in the third hour of performing physical work), complained of mild painful sensations in his right knee joint. These sensations lasted for a few cycles of the muscular work, and then disappeared.

After ascent to 11,000 m, pronounced painful decompression symptoms developed very rapidly (toward the 15th minute) and

were localized in the same region. This case suggests the idea that formation of gas bubbles that cause painful sensations can, in individual cases, occur under the condition of muscular /288 work even at an altitude of 5,000 m. Evidently, without physical work this possibility is practically excluded.

In the next chapter, material will be presented from 200 experiments involving a stay of examinees (a total number of 38 men) at an altitude of 5 km for from 10 to 24 and even 48 hours without performing physical work; this material contains no case of complaints of any decompression symptoms not only of a painful, but even of any other kind (sensations of discomfort, etc). Important, moreover, is the fact that the examinees breathed at the altitude not pure oxygen, but a 40 and 45% mixture of it with nitrogen. Even earlier, E. V. Bondarev, A. M. Venin, G. I. Gurvich et al [1969] carried out 100 similar experiments with the same results. Thus, if it is even permissible, on the basis of the case referred to, to conceive of the possibility of decompression symptoms arising at an altitude of 5,000 m, then we can do so only under the condition of a physical load.

In testing different desaturation conditions, we endeavored in one experiment to combine it with increased temperature of the surrounding medium (+30°C at a humidity of 60%), and in another, with gradual reduction, which was stretched out over the entire six hours, of the pressure in the chamber down to an altitude of 11,000 m. In both cases, the examinees developed pronounced decompression disorders. Table 18 sums up the results of all the just-mentioned experiments with special desaturation conditions and ascent to an altitude of 11,000 m.

As can be seen from the table, these experiments did not justify our hopes to find some additional conditions which might clearly increase the effectiveness of desaturation. This number of experiments, finally, is insufficient to draw categorical conclusions about the nature and extent of the influence of the tested conditions on the effectiveness of desaturation. It is probable, for example, that by performing very strenuous muscular work accompanied by high indices of pulmonary ventilation and blood circulation, desaturation from nitrogen would be more complete and, perhaps, somewhat more effective from the standpoint of preventing altitude decompression disorders; it is possible that increasing the number of experiments with increased temperature or with ascents stretched out in time would disclose some relative advantages of desaturation under such conditions. But neither heavy and prolonged physical work nor conditions that promote development of hyperthermia before the astronaut's emergence into space is acceptable. The important thing to be stressed is that reduction of the number of cases of decompression disorders for one or another version of

desaturation is an experimental result that can have no practical application. Prophylactic measures to prevent decompression disorders when astronauts emerge into space must preclude the possibility of these disorders developing. From this standpoint, the heuristic experiments undertaken disclosed no additional prospects and only strengthened the conclusion /289 ensuing from the experiments of A. V. Kuznetsov, I. P. Poleschuk and their coauthors that the method of desaturation of the organism from nitrogen by means of oxygen breathing is insufficiently effective for decompression disorder prophylaxis under space flight conditions.

TABLE 18. RESULTS OF EXPERIMENTS WITH DESATURATION UNDER DIFFERENT CONDITIONS

Experimental conditions	Number			Time of appearance of altitude decompression disorders
	Experiments	Examinees	Altitude decompression disorders (pains)	
Desaturation for 5, 6 and 8 hours in an oxygen-filled airtight suit	15	8	11	10 min - 2 h 30 min
Desaturation for 6 h: while performing muscular work on the ground	5	4	5	40 min - 2 h 05 min
while performing muscular work at an altitude of 5 km	3	2	2	15 min - 1 h 30 min
incident to increased temperature	1	1	1	10 min
incident to slow 6 h ascent to 11 km	1	1	1	2 h 05 min

Nor did increasing still more the duration of desaturation and finding that time limit for which there will be no decompression disorders make any practical sense inasmuch as the 6- and 8-hour periods of oxygen breathing that were tested (with

unsatisfactory results) met neither technical nor physiological requirements, nor were they satisfactory from the point of view of organizing space walks. Consequently, the need arose to work out experimentally some other means of decompression disorder prophylaxis in astronauts emerging into space. The following chapter is devoted to this question.

But here, in order to put an end to the question of desaturation, it is necessary to evalute its effectiveness and the possibility of its practical utilization for decompression disturbance prophylaxis in the case of the lower degrees of rarefaction employed in spacesuits. To be sure, increasing the pressure in spacesuits, insofar as this has a negative effect on the astronaut's mobility and encumbers his actions, is /290 undesirable, and this method of decompression disorder prophylaxis is to a considerable extent in the nature of a compromise and a palliative. But from the theoretical and practical points of view, it is necessary to check the effectiveness of desaturation for the prevention of decompression disturbances at lesser degrees of rarefaction. Not wishing to digress from the extreme (permissible for the organism's oxygen provision) degrees of rarefaction in spacesuits or from the task of radical solution of the problem of decompression disorder prophylaxis incident to space walks, we did not work out the practical side of this question. But for the purpose of analysis of the conditions under which altitude decompression disturbances arise, we carried out experiments with different desaturation times, transition to altitudes of 7,000 and 8,150 m and the usual program for the stay of examinees under these conditions (six-hour exposure while performing muscular work). The results of these experiments reduced to the following:

Conditions	Number of experiments	Number of examinees	Number of cases of altitude decompression disorders (pains)
Altitude 7,000 m, desaturation 20 min	12	11	7
Altitude 7,000 m, desaturation 1 h	21	8	6
Altitude 8,150 m, desaturation 20 min	19	14	13
Altitude 8,150 m, desaturation 40 min	8	8	5
Altitude 8,150 m, desaturation 1, 2, and 4 hours	5	5	5

The adduced figures again attest to the insufficient effectiveness of desaturation even as applied to these comparatively low altitudes, under the condition of prolonged exposure and a physical load. The 20-minute periods can be left out of account; they were employed not for the purpose of preventing bends symtoms, but for the purpose of weakening possible aeroembolic phenomena. But the fact that, after an hour of desaturation at an altitude of no more than 7,000 m, i.e., under the pressure that is regarded as the upper limit for the working pressure in a spacesuit (308 mmHg), decompression disturbances developed in almost one-third of the cases, compels us to take a pessimistic stand with regard to practical utilization of desaturation.

The adduced data do not, of course, settle the question. A study by A. M. Venin and his coauthors [1973] was devoted to study of this question. In investigations on people, the authors checked the effectiveness of desaturation from nitrogen by means of breathing either pure oxygen or gaseous mixtures containing 40-45% O_2 and 60-65% N_2 at an altitude of /291
4,500 m (a total pressure of 430 mmHg) with a view to a subsequent stay at altitudes of 7,000-10,000 m (a total pressure of 308-200 mmHg). The examinees either were in the pressure chamber atmosphere and breathed oxygen from an instrument or were dressed in altitude suits in which the indicated pressure was created; the degree of rarefaction of the air in the pressure chamber, however, reached much higher values. On the basis of different versions of the experiments (duration of decompression and ascent conditions), the authors arrived at two fundamental conclusions. In the first place, breathing O_2 for two hours under a pressure of 430 mmHg rather effectively prevents altitude decompression disorders from developing incident to a subsequent 5-6-hour exposure at an altitude of 7,000 m while performing strenuous physical work. In the second place, breathing O_2 for five hours under the indicated pressure or ten hours of desaturation under this same pressure by means of breathing a mixture of 40% O_2 and 60% N_2 prevents decompression disorders from developing at an altitude of 10,000 m (as well as for a five-hour exposure and strenuous physical work). Though there was not a single case of decompression disorders out of 23 experiments conducted on 15 examinees breathing pure O_2, they did develop in one examinee out of 12 experiments using the indicated mixture.

As long as no other, more acceptable methods of decompression disorder prophylaxis have become a part of space flight practice, these data can serve as practical guidelines for preventing decompression disturbances in this manner.

But, on the whole, they are not very comforting. In the first place, the desaturation times are rather long, which is a

disadvantage for practical applications. Even for the highest working pressure in a spacesuit (0.4 atm -- 308 mmHg), under which an astronaut's working efficiency is considerably hampered, they amount to two hours; and for a pressure of 200 mmHg (0.27 atm), which does not yet attain the lower limit of the working pressure, they equal five hours. In the second place, and this is even more important, the given times may not be any guarantee against development of altitude decompression disorders when using the minimum permissible values of the working pressure in a spacesuit (170-180 mmHg). Under real conditions, when an astronaut is working in empty space, it may become necessary to change over to this pressure in the spacesuit. In this case, decompression disorder development may pose a threat to accomplishment of the astronaut's work and to his well-being.

Results of experiments involving latent forms of decompression disorders induced by means of additional ascents to an altitude of 11,000 m

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Taking into account the above-described possibility of latent forms of decompression disturbances being clearly brought out in animals by means of ascents to an altitude after their emergence from under safely increased pressures, we began systematically to use a similar procedure in investigations on people in all those cases where the onset of decompression disorders was studied incident to lesser degrees of rarefaction than the maximum permissible rarefaction in spacesuits. We are talking about experiments carried out by I. P. Poleschuk and V. P. Katuntsev with the participation of R. T. Kavakova, S. I. Tokareva, N. Yu. Leont'yeva et al., in which examinees had to perform the usual six-, and sometimes four-hour program using the above-described physical load at altitudes from 6,000 to 9,000 m.

In most of these experiments, the results of which have already been partially described earlier, desaturation was not employed; in some of them, oxygen was first breathed for 20, 40 and 60 minutes. In those experiments where acute painful sensations arose in the examinees, they were lowered to the ground. For the time being, these experiments will be left out of the discussion. Within the framework of inducing latent forms of decompression disorders, other cases are of interest. These are, in the first place, those experiments in which relatively weak, tolerable painful sensations arose which did not require interrupting the experiment; the examinees continued to perform the experimental program, sometimes skipping individual cycles of the muscular work, and with the passage of time, their painful symptoms disappeared. In the

second place, these are those experiments in which the examinees in the course of a six- or four-hour stay at altitudes ranging from 6,000 to 9,000 m did not at all develop any painful sensations or any other signs of decompression disorders in the form of a feeling of heaviness, discomfort in one or another region of an extremity, etc.

In all of these experiments of the first, as well as the second, category, after terminating the planned experimental program, the examinees were subjected to additional rapid ascent to higher altitudes, up to 11,000 m. If we take the first category of experiments with additional ascents (reascents), when in the course of performing the main experimental program the examinees showed weak decompression pains which then disappeared, incident to the final ascent these pains in the overwhelming majority of cases reappeared. These facts are not at all unexpected or new. As early as 1947, Rodbard studied the appearance of altitude decompression pains, ascertained their disappearance after descent to the ground, and then undertook reascents after different periods of time. He found that for comparatively short intervals between first and second /293
ascents, bends relapses are the rule; with an increase in the periods, their number decreases, and after a three-hour interval they no longer appear. The constant renewal, during reascent to an altitude, of painful symptoms that had disappeared incident to descent was noted in the above-discussed experiments of L. V. Kalyuzhnyy and his coauthors directed at studying the functions of the central nervous system incident to decompression. Individual cases of recidivism of bends symptoms incident to repeated ascents are mentioned in the study of A. M. Genin, I. N. Chernyakov and their coauthors.

Thus, the renewal of painful decompression symptoms incident to reascents following closely upon the heels of the initial action of the rarefaction that caused these symptoms is a fact of rather frequent occurrence and in essence quite understandable. It is well-known from theoretical considerations and from the practice of therapeutic recompression of divers that the gas bubbles that cause painful symptoms are not liquidated immediately when the pressure is increased; for their resolution and disappearance a certain more or less protracted time is necessary. Initially, an increase in pressure leads to disappearance of painful sensations because of a shrinking and decrease in volume of the bubbles; therefore, incident to reascent effected after some not too long time after initial ascent, these gas bubbles begin to grow at once and lead to renewal of the symptoms.

Of considerably greater interest is the second category of experiments, in which no decompression symptoms were noted

when the main experimental program was accomplished. It turned out that under these conditions, too, reascents effected at the end of the main experimental program lead to the onset of pronounced, and often even very severe, painful decompression symptoms; especially interesting are those cases where these symptoms already arise and develop rapidly in the course of the ascent. Here, then, we have absolutely the same effect that was detected in the experiments with decompresion disorders induced in dogs by raising them to an altitude after their emergence from under increased pressures, which did not lead to development of any decompression disturbances.

This effect in the investigations on people that we are now considering, as well as in the experiments on dogs that we discussed earlier, can be interpreted in only one way. It directly indicates that there had already been formed in the organism gas bubbles which, however, either in virtue of their insufficient volume or in virtue of the insufficient internal pressure in them, have not yet led to development of painful sensations; incident to reascents, these "silent" gas bubbles immediately make themselves known and cause pronounced, and sometimes very severe, pains. Besides its theoretical interest,/294 this fact, in our opinion, is of great practical importance. It compels us to ponder how valuable the desaturation methods proposed by different authors are as safe and effective methods incident to transition to intermediate or low degrees of rarefaction (altitudes of 9,000-7,000 m). The results of the just-described experiments give every reason to suppose that in the human organism, incident to complete external safety and in the absence of any subjective signs of decompression disorders, there exist "silent" gas bubbles. In the event of a forced reduction of the working pressure in a spacesuit, which may under practical conditions be dictated by the necessity of ensuring the astronaut's greater mobility, these "silent" gas bubbles may manifest themselves at once and lead to severely impaired efficiency.

Worthy of attention are also other cases of onset of painful symptoms incident to reascents, when in the process of performing the main experimental process, such symptoms were absent. These cases are characterized by the fact that the pains arise not in the course of the ascent itself, but incident to reaching the maximum permissible altitude, 11,000 m, their appearance in such cases being noted, as a rule, in a comparatively short time, countable in minutes, after reaching this maximum permissible altitude. Here we can think either that the "silent" gas bubbles were very small in size before reascent and rapidly grew incident to additional rarefaction or that almost all of the conditions for their onset had already been laid down before reascent. From the practical point of view, such cases differ but little from those examined above inasmuch as incident to

them, a real threat of impairment to the astronaut's efficiency is also created incident to forced transition to a lower pressure in his spacesuit.

Besides the practical considerations we have just been talking about, these results point to the expediency of using the method of inducing latent forms of decompression disorders by means of final reascents to maximum permissible altitudes after a preliminary stay of the examinees under conditions of more moderate degrees of atmospheric rarefaction. They are convincing evidence of the fact that in order to place on a firm foundation and work out effective means of decompression disorder prophylaxis in astronauts, we must be guided by the lowest working pressures in spacesuits.

Summing up what has been set forth in the present chapter, it is necessary, first of all, to note that altitude decompression disorders constitute an obvious and considerable threat to man incident to his transition from normal pressure without desaturation to all of those values of increased pressure that make up the range of working pressures in a spacesuit. Indeed, the facts adduced above show that incident to ascending without /295
desaturation even to an altitude of 7,000 m (308 mmHg) and staying there for many hours while performing physical work, decompression disorders arise in more than half of the cases. Sometimes they abate spontaneously and disappear, but very often they progress to extreme proportions and lead to severely impaired efficiency. It may be that these results are to a considerable extent determined by the nature and intensity of the muscular work we chose. In the studies of several authors on this question, somewhat different results were obtained, and the task of future investigations will be to elucidate what this difference consists in. But the facts adduced above can in no way be left out of account, and these facts point directly to the fact that under the experimental conditions we used, decompression disorders incident to rarefaction corresponding to an altitude of 7,000 m without any prophylactic measures, constitute a real threat to astronauts. If we go over to the lower pressures that may be used in spacesuits, this threat, according to our data as well as to the results of other investigators, increases steadily.

It follows from the material expounded above that incident to a man's transition to considerable degrees of rarefaction without preliminary desaturation, in the pattern of decompression disorder development in the first period, an important part is undoubtedly played by aeroembolism. According to all of the data, it does not, as a rule, attain that intensity which is characteristic of cases of rapid decompression from under increased pressures. But as the general, most typical mechanism of initial development of decompression disorders, it remains

valid for altitude conditions, too. The most noteworthy result of the investigation presented above is, in our opinion, those facts which attest to the changes in function and state of the nervous system that set in even before development of pronounced decompression symptoms of the bends type. These facts permit us to consider it probable that the general condition and efficiency of an astronaut going over to the low pressure conditions in his spacesuit may be changed and to a certain extent detected even when he does not subjectively feel any of the clear discomfort that is characteristic of decompression disorders. These same facts are directly connected with the data on formation of "silent" gas bubbles incident to atmospheric rarefaction of one degree or another or, in other words, with data on latent forms of decompression disturbances.

Experiments using desaturation from nitrogen by means of oxygen breathing as a means of decompression disorder prophylaxis in astronauts did not, on the whole, yield reassuring results.

In searching for effective desaturation times as applied to /296
the lowest values of pressure in a spacesuit (170 mmHg), the matter already came to 6 and 8 hours, and even these intervals proved to be insufficient to completely prevent decompression disorders. These periods, as has already been noted, do not properly meet practical requirements either from the technical or the physiological point of view. The somewhat shorter desaturation times determined by other researchers, which are effective, according to their data, for decompression disorder prophylaxis at lesser degrees of rarefaction, also prove to be rather considerable. But the main point, as has been stressed, does not even consist in this, but in the fact that the absence of pronounced decompression symptoms incident to using such desaturation for intermediate and higher spacesuit pressures still gives no guarantee that "silent" gas bubbles will not be formed in the organism. Should it become necessary to use lower (extreme from the standpoint of oxygen provision) pressures in the spacesuit, these "silent" gas bubbles may lead to serious complications of the astronaut's activity and create threatening situations.

It ensues from all of the foregoing that desaturation of the organism from nitrogen by breathing oxygen under normal pressure conditions is not a reassuring or promising way of ensuring practically acceptable and effective decompression disorder prophylaxis in astronauts.

Proceeding from this, our investigations connected with the present problem took a different direction, which is what the following chapter will be about.

CHAPTER 15

ARTIFICIAL GASEOUS MEDIA WITH A REDUCED TOTAL PRESSURE AND A NORMALIZED PARTIAL OXYGEN PRESSURE AS A MEANS OF DECOMPRESSION DISORDER PROPHYLAXIS UNDER SPACE FLIGHT CONDITIONS

As has already been pointed out, the very serious problem of choosing optimum artificial gaseous media for spacecraft cabins is still far from solved. This is borne out by the fact that in recent years many researchers have kept returning to discussion of this problem in the scientific literature.

This problem is complicated by the fact that in solving it, /297 it is necessary to take into account not only a great number of purely technical circumstances, which it is not always easy to unravel, but also a great number of physiological and medical considerations. I shall not treat all sides of this problem but only that aspect which was the subject of our investigations. It is a question of selecting such an artificial gaseous medium for spacecraft cabins as will not only meet the vital requirements of an artificial atmosphere, but will also solve the problem of artificial decompression disorder prophylaxis in astronauts when they change into their spacesuit and engage in activity outside the spacecraft.

First of all, it is necessary to note that the formulation of this problem is not new. The question of artificial gaseous media that would reduce the danger of decompression disorders and the noxious consequences of explosive decompression has been raised in a general manner in a great number of studies. More than that, E. V. Bondarev, A. M. Genin and their coauthors [1969] tested experimentally an artificial gaseous medium especially designed to prevent altitude decompression disorders. The authors conducted 100 experiments on examinees who for ten hours breathed through a mask a gaseous mixture consisting of 45% O_2 and 55% N_2 under a total altitude-chamber pressure of 405 mmHg. After this, the examinees were subjected to the action of a pressure of 170 mmHg while breathing pure O_2 for four hours without performing systematic physical work. In not one of the 100 experiments did the authors note the onset of altitude decompression disorders in the examinees under the indicated conditions, on the basis of which they concluded that such a gaseous medium might be promising for decompression disturbance prophylaxis as applied to space flight conditions.

But absent from the investigations of these authors was the condition that is important and obligatory for the activity of an astronaut in a spacesuit, namely, a systematic physical load, whose provocative role in respect of altitude decompression disorders we have already talked about. Moreover, it might have

been desirable to prolong somewhat the staying time in the rarefied atmosphere and the real activity of an astronaut in space.

After all of our earlier investigations had convinced us of the relatively little promise of desaturation by means of oxygen breathing incident to normal pressure for altitude decompression disorder prophylaxis on space flights, the main efforts of our team were directed at experimental study and development of a gaseous medium with a reduced pressure as a means of preventing decompression disturbances in astronauts.

In our experiments, we used the same gaseous medium that /298 was used in the study by A. M. Genin and his coauthors, i.e., an artificial nitrogen-oxygen atmosphere with a total pressure of 405 mmHg, consisting of 45% O_2 and 55% N_2. But into our investigations we immediately introduced two new conditions: firstly, performance of systematic physical work (what this consisted in has been discussed in detail above) by the examinees in the final stage of the experiment under a pressure of 170 mmHg and, secondly, lengthening of the staying time in the rarefied atmosphere up to six hours. In all subsequent investigations, we firmly and persistently adhered to these two conditions. This--in particular the use of rarefaction to 170 mmHg--we were induced to do by the consideration that the prophylactic measures we worked out had to guarantee the absence of decompression disorders under the most drastic conditions. If they could meet these conditions, then they would clearly be suitable and effective under less drastic conditions, i.e., for the higher working pressures in a spacesuit.

In initial experiments conducted jointly with A. M. Genin, I. N. Chernyakov et al, we kept the previous ten-hour staying time of the examinees in the indicated gaseous medium in the first stage of the experiment, but into the second stage we introduced a physical load and lengthened exposure to six hours. In three out of eight such experiments, pronounced decompression disorders were detected. Here, as in all subsequent versions of the experiments, we did not strive to increase the number of experiments with similar results. Even the isolated negative results attested to the fact that the method of transition to a pressure of 170 mmHg that we used (under the condition of six-hour exposure and muscular work) is not absolutely safe.

Consequently, it was not possible to consider this method as being sufficiently full-fledged, effective and suitable for practice and we had to go on to look for another method. In the next series of experiments, the staying time of the examinees in the indicated gaseous medium in the first stage of the experiment was lengthened to 14 hours. Twenty experiments were conducted on 20 examinees. In two cases, pronounced decompression disorders were once again recorded, which developed in one

experiment 1 hour 7 min, and in another, 3 hours 50 min after ascent to an altitude of 11,000 m.

The next series of investigations (ten experiments on ten examinees) with 16-hour exposure in the indicated gaseous medium did not yield the desired results, either: in two cases (the fourth and fifth hours of staying at an altitude of 11,000 m) pronounced altitude pains were noted, which obliged us to interrupt the experiment.

Thus, in this stage of the investigations, the results also proved to be unfavorable, and in this respect they approximated, as it were, the results of the experiments with desatuation by means of oxygen breathing. But we had to approach evaluation of these two possible ways of decompression disorder prophylaxis (breathing pure O_2 under ordinary pressure and staying in a nitrogen-oxygen gaseous medium under reduced pressure) in different ways. If very prolonged breathing of O_2, as has already been noted, is unacceptable in practice for technical and physiological reasons, lengthening of a man's staying times in the gaseous medium we chose is practically quite feasible. On these grounds it is conceivable that such a gaseous medium will be the atmosphere of the entire spacecraft cabin. As will be shown in greater detail below, in virtue of its fundamental physiological properties (normal oxygen provision of the organism, absence of any pronounced unfavorable effects on other physiological parameters), it is suitable for a man's prolonged stay in it. This gave us grounds to continue the investigation with increased staying times in the gaseous medium in question.

The staying time in the gaseous medium in the first stage of the experiment was increased to 18 hours. Experiments were conducted on 31 examinees. At first, in conducting the more than 30 experiments, the results proved to be reassuring: no cases of decompression disorders were encountered. But then they began to appear. The experiments had to be continued and their number was increased to 74. In 12 experiments on seven examinees, clear painful symptoms of altitude decompression disorders were recorded. The times of their appearance varied greatly. In one case, they appeared in the eighth minute of staying at the altitude.

The given case, however, was an exception because in all of the remaining experiments, the times ranged from 1 h 05 min to 3 h 40 min.

The degree of manifestation of the painful sensations and the rapidity of their increase in intensity in individual examinees were different. But in all 12 cases, they reached such an intensity that it was necessary to interrupt the experiments and effect descent to the ground. Thus, even 18-hour exposure in the gaseous medium with a total pressure of 405 mmHg, consisting

of 45% O_2 and 55% N_2, did not yield the desired results and proved to be insufficient for the prevention of altitude decompression disorders. But the latter appeared not so frequently and this encouraged us to hope that we were approaching that temporal bound at which desaturation of the organism from nitrogen in the indicated gaseous medium can turn out to be effective for decompression disorder prophylaxis. These hopes were confirmed in the final series of experiments using the /300 given gaseous medium.

Exposure was increased to 24 hours; 50 experiments were conducted on 19 examinees. In no case were pronounced painful decompression symptoms detected. A certain exception was only one experiment, in which the symptoms arose in a very mild form and were expressed in mildly painful sensations.

This experiment refers to an examinee who was distinguished by high sensitivity to decompression disturbances. But among the indicated 19 examinees, there were also others who were little resistant to decompression disorders, so that the obtained result, attesting on the whole to the sufficiently reliable prophylactic effect of the utilized gaseous medium incident to a 24-hour stay in it, must be considered effective for people with different, including low, resistances to decompression disorders.

Thus, in the just-described series of experiments, the desired result was finally achieved. The facts showed that a gaseous medium with a total pressure of 405 mmHg and consisting of 45% oxygen under the condition of a man's staying in it no less than 24 hours can be regarded as a sufficiently reliable means of decompression disorder prophylaxis upon transition of astronauts to the lower working pressure in their spacesuit (170 mmHg) under the condition of a prolonged physical load. This was the main practical result of our investigations.

In putting forward this recommendation, we based ourselves on the above-mentioned concrete parameters of the gaseous medium that were worked out experimentally. At the same time, proceeding from the multitude of data obtained as a result of our investigations, it was possible in principle to expand this recommendation. In other words, logical was the general conclusion that a two-component nitrogen-oxygen gaseous mixture with a moderately reduced total pressure and a normalized partial O_2 pressure is a promising prophylactic means of preventing decompression disorders on space flights. In that case, some modification of the parameters of this gaseous medium (total pressure, oxygen and nitrogen content) as a function of the real practical conditions of its use should be regarded as the partial concretization of this principle of decompression disorder prophylaxis as applied to the conditions in question.

After carrying out these investigations, we were confronted with the theoretical question of how to evaluate the obtained results. Here it was possible to make two surmises: firstly, it could be supposed that a practically complete equilibration of the organism with the changed gaseous medium takes place in a period of 24 hours, i.e., that the partial pressure of the /301 nitrogen in the organism becomes equalized with the surrounding pressure corresponding to 55% of nitrogen at a total pressure of 405 mmHg (equal to 223 mmHg). Then it could be concluded that precisely this level of partial pressure of the nitrogen in the organism is safe in respect of development of decompression disorders at an altitude of 11,000 m. Secondly, it could be imagined that a period of 24 hours is still insufficient for establishment of complete gaseous equilibrium, and that in this period the pressure of the nitrogen in the organism drops to values that still exceed the partial pressure of the nitrogen in the surrounding medium but are already safe in respect of altitude decompression disorders.

In order to approach elucidation of this question, it was expedient to check how a gaseous medium with the same total pressure of 405 mmHg but a partial oxygen pressure reduced by 20 mmHg and a correspondingly increased partial nitrogen pressure (40% oxygen and 60% nitrogen) would behave in respect of decompression disorder prophylaxis. To this end, we carried out some series of experiments with different staying times in a gaseous medium consisting of 40% oxygen and 60% nitrogen. Seven examinees were raised to the altitude with a 12-hour exposure, and all of them developed altitude decompression disorders. Twenty-four experiments were carried out with a 16-hour exposure -- in seven of them, decompression disorders were detected; upon increasing the staying time in the gaseous medium to 24 hours, they appeared in three of the four examinees participating in these experiments. And finally, in one out of two experiments with a 48-hour exposure, decompression disturbances also arose.

It can be seen from these results that a gaseous medium with a total pressure of 405 mmHg and consisting of 40% oxygen and 60% nitrogen, in contrast to a gaseous medium with the same pressure but consisting of 45% oxygen and 55% nitrogen, does not prevent decompression disorder development at an altitude of 11,000 m even incident to very prolonged periods, including 48 hours, of a man's preliminary stay in this medium. Whence it follows that the difference in the partial nitrogen pressures of the two indicated media, which amounts in all to only about 20 mmHg, is of very definite significance for the onset of decompression disorders -- though it should be noted that for a rarefied atmosphere, the level of the partial pressure of the nitrogen in the organism that is dangerous in respect of decompression disorder development must be regarded, if one may so express it, as the "nitrogen background" on which the

physically dissolved oxygen and carbon dioxide are superposed. The total degree of supersaturation of the tissues with all these gases determines, as has already been noted, the possibility of postdecompression gas-bubble formation.

Of the two variants of gaseous media with a total pressure /302 of 405 mmHg that we checked, only the medium containing 45% oxygen and 55% nitrogen can be recommended for reliable decompression disorder prophylaxis at an altitude of 11,000 m.

In analyzing and interpreting the experimental data, I. P. Poleschuk and V. P. Katuntsev noticed the following regularity. When they compared experiments with ascents to altitudes of 6 and 7 km above the ground with experiments in which examinees spent a long time in the above nitrogen-oxygen media under a pressure of 405 mmHg and were then raised to an altitude of 11,000 m, they obtained the dependence that is depicted graphically in Fig. 74. This is a plot of the initial partial pressure of the nitrogen in the organism under the condition of sufficiently complete equilibrium of the latter with the initial gaseous medium against the total pressure after transition to the rarefied atmosphere. The upper straight line (the solid line in the figure) denotes those ratios between the indicated values at which decompression disorders do not arise. This straight line rests on two points that were thoroughly checked experimentally. The right (upper) point corresponds to experiments involving transition from ordinary pressure to an altitude of 6,000 m without desaturation. The left (lower) point corresponds to experiments involving a 24-hour stay of the examinees in a gaseous medium with a 45% oxygen and 55% nitrogen content under a total pressure of 405 mmHg and subsequent transition to an altitude of 11,000 m. In both cases, as has already been pointed out, decompression disorders were absent. Hence it can be concluded that the straight line joining these points reflects in the pressure range presented in the figure the safe ratios of the partial pressure of the nitrogen in the organism before decompression to the total pressure after decompression. The lower straight line (broken line in the figure) reflects dangerous ratios between these values. It, too, rests on two points: the right (upper) point corresponds to experiments involving transition from normal pressure to an altitude of 7,000 m and the left (lower) point corresponds to experiments involving a stay of examinees in a gaseous medium containing 40% oxygen and 60% nitrogen (a total pressure of 405 mmHg) and their transition to an altitude of 11,000 m. In both versions of these experiments, decompression disorder development took place. In evaluating these curves, we can consider it probable that the entire region situated above the upper straight line corresponds to safe ratios between the two fundamental values that determine decompression disorder development:

P_{N_2}/P_{total}. The region situated below the lower curve corresponds to dangerous ratios between these values. There can be no doubt that the straight lines in question require additional, experimentally well-founded, "points of rest" before they can acquire the character of an incontestible regularity. But on the basis of all of the material that we have analyzed and of data obtained in current work, we can consider it pretty probable that such a regularity will finally be confirmed. It is of great practical interest inasmuch as on its basis we can determine beforehand the danger or safety of any concrete methods of transition from one or another initial gaseous medium to a rarefied atmosphere. In evaluating the nomogram from the theoretical point of view, it is easy to see that it closely approximates those notions of the coefficient of maximum permissible supersaturation of the organism with nitrogen that are widely utilized in diving practice. /303

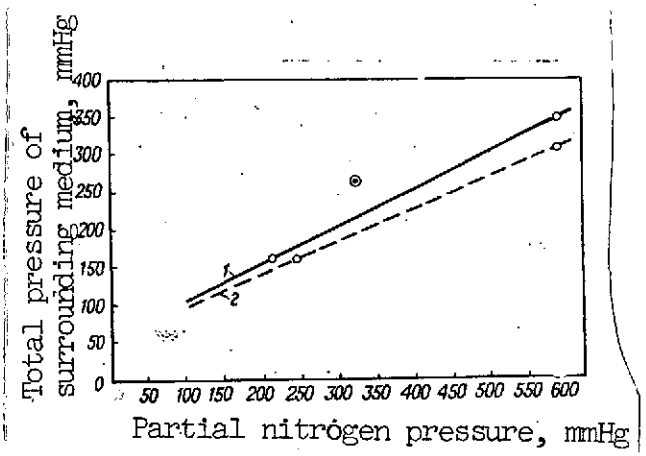

Fig. 74. Dependence between initial pressure of nitrogen in organism and total pressure of surrounding medium after decompression, reflecting probability of altitude decompression disorder development. 1 -- ratios between values in question that are safe in respect of altitude decompression disorder development; 2 -- ratios between these values, at which decompression disorders arise. Point in circle corresponds to ratio of values in question incident to transition from gaseous medium with pressure of 540 mmHg (40% oxygen, 55% nitrogen) to pressure of 258 mmHg (on assumption of sufficiently complete equilibrium of organism with indicated gaseous medium).

It is not, of course, a question of a strict agreement of figures for the two cases, but only of a matter of principle; in other words, of general points of departure from which it might be possible to proceed in determining safe decompression conditions.

In speaking of artificial gaseous media for spacecraft cabins, it must not be forgotten that, in principle, as oxygen diluents can be used not only nitrogen, but also other inert, as it is customary to call them, gases. In a number of investigations devoted to the problem of an artificial gaseous atmosphere for spacecraft cabins, helium in particular has been considered. Here I shall not reproduce the different technical as well as physiological arguments advanced by authors for and against using helium as an oxygen diluent in artificial gaseous media. In our current investigations, helium is of interest in connection with the problem of decompression disorder prophylaxis. /304

In spite of the fact that at the present time, the possibility of using helium in spacecraft cabins is mostly regarded as problematical, we thought that space physiology ought to accumulate many-sided materials concerning one or another concrete problem. That is why our team also carried out experiments using helium. The investigations were conducted, under the immediate scientific guidance of A. G. Dianov, by him and his laboratory coworkers. The purpose of the investigations was to achieve, by means of a prolonged stay of examinees in a helium-oxygen atmosphere, complete replacement of the nitrogen in the organism's media by helium and then to trace decompression disorder development under the action of a rarefied atmosphere. The program of the final stages of the experiments was the very same as that in all of our previously described experiments. The rarefaction corresponded to an altitude of 11,000 m (170 mmHg), exposure amounted to six hours and the above-mentioned physical load was used. But the first stage of the experiments consisted in placing the examinees for 24 hours (and sometimes 48) in an altitude chamber filled under ordinary pressure with a gaseous medium consisting of 21% oxygen and 79% helium. These periods, according to all of our data, ensured practically complete elimination from the organism of nitrogen and its just as complete saturation with helium.

It should be noted that no studies can be found in the literature in which such conditions were adhered to.

After the examinees had stayed 24 hours (sometimes 48) in a helium-oxygen medium, more or less prolonged desaturation of the organism from helium was effected by breathing pure oxygen, after which the examinees were raised to an altitude of 11,000 m. Desaturation time varied from experiment to experiment, and the main purpose of the experiments was to find those times which would be effective from the standpoint of decompression disorder prophylaxis and to compare them with the corresponding times when air is used (incident to saturation of the organism with nitrogen). In this connection, all of the results of the investigations with an initial air mixture that have been set forth above were sufficiently reliable and constituted extensive material for such a comparison.

The investigations to a considerable extent yielded new and unexpected results which, in our opinion, are of interest from the theoretical, as well as practical, point of view. It turned out that the effective desaturation times when a helium-oxygen atmosphere was used were incomparably shorter than when an air atmosphere was used.

On the basis of the rather extensive material that has already been obtained, it can be concluded that one-hour desaturation /305
practically quite completely prevents decompression disorder development at an altitude of 11,000 m incident to six-hour

exposure while performing muscular work. In current experiments, data are being accumulated to the effect that, as a rule, even 30-minute exposure is sufficiently effective. On theoretical grounds and from diving practice, more rapid desaturation of the organism from helium was to be expected when breathing oxygen as compared with its desaturation from nitrogen. Helium, as is well-known, possesses a high diffusibility, and this property that it has is taken into account in diving practice and is utilized, in particular, in implementing the principle of alternate supply of different respiratory mixtures at different stages of diving.

However, on the basis of all of the above theoretical considerations and for practical reasons, we did not expect such sharp differences in safe times of desaturation of the organism from helium and nitrogen as were detected in the experiments of A. G. Dianov and his coworkers. There can be no doubt that this question requires further treatment in connection with its theoretical interest, as well as from the standpoint of the possible future practical utilization of helium for decompression disorder prophylaxis under space flight conditions.

CHAPTER 16

SOME DATA ON PHYSIOLOGICAL ANALYSIS OF THE DISTINCTIVE FEATURES OF ALTITUDE DECOMPRESSION DISORDER DEVELOPMENT

There can be no doubt that in order to consolidate reliable and effective measures of altitude decompression disorder prophylaxis as applied to space flight conditions, in-depth physiological analysis of altitude decompression disturbances is absolutely necessary, as well as elucidation of their external and internal conditions and their mechanism of development. Without such an experimental, analytical and theoretical basis it is inconceivable that this problem could be treated in a scientifically and practically fruitful manner. This is all the truer, as the altitude side of the problem of decompression disorders in connection with the requirements of astronautics has brought out a number of unexpected, extremely interesting and important peculiarities concerning the development of these distinctive functional disturbances in a rarefied atmosphere. /306 Analysis of these distinctive features and their comparison with material emanating from the domain of diving physiology and caisson operations can shed light on a number of cardinal problems of decompression disorders as a whole and can serve to perfect methods for their prevention and to ensure man's safety in all spheres of activity attended by decompression effects.

In connection with this, in our current investigations devoted to altitude decompression disorders, a considerable place

is occupied by experimental and analytical studies. Of some of them, in particular the electroencephalographic investigations of the state of the central nervous system that have been carried out by L. V. Kalyuzhnyy and his coauthors, we have already spoken above inasmuch as these investigations, though they are profoundly analytical in character, in the way they were carried out and in their practical purposes are closely akin to the main line of research examined in the preceding chapters.

In the present chapter we shall report on the results of a number of other studies that we carried out on physiological analysis of altitude decompression disturbances.

Chronic experiments conducted on animals in order to induce altitude decompression disorders

The possibility of observing the development of altitude decompression disorders in animals is very tempting. It would permit us to some extent to curtail investigations of people, to carry out experiments involving study of all of the dynamics of decompression disorders, right up to their most serious manifestations, and to determine the outcome of their development incident to the prolonged action of rarefaction on the organism. Here, however, arise difficulties connected with the fact that animals are more resistant to decompression disturbances than man. The closest to man in their resistance to decompression disorders, as has already been mentioned, are dogs and rabbits. But even they, as is well-known to specialists in altitude physiology, seldom show decompression disorder symptoms even at those extreme altitudes at which serious hypoxic phenomena can still be avoided by breathing oxygen. I can refer to the great experiment of Professor M. P. Brestkin's laboratory and his personal experience in carrying out chronic experiments on dogs involving their ascents in a pressure chamber to altitudes ranging from 11 to 13.5 km while breathing oxygen through a mask. In these experiments, which had different purposes (mostly, investigation of the respiration, blood circulation and gaseous composition of the blood under these conditions and study of /307
respiration under an overpressure), the experimentors had to be in the chamber together with the dog (to take blood samples, check the position and airtightness of the mask, etc) very often for a rather long period of time (1-1.5 hours). As a rule, preliminary desaturation of the experimentor (brief though it usually was) prevented him from developing decompression disorders. But the experimental dogs did not undergo desaturation, and they were given oxygen to breathe either from the start of ascent or upon reaching the altitude. In spite of this, in these experiments they displayed decompression symptoms very seldom.

Of the many dozen experiments of this kind that I carried out, I can recall only two cases of clear bends symptoms

developing in two husky and very well-fed dogs at an altitude of 13 km.

Thus, the prospects of inducing altitude decompression disorders in dogs were not promising. Nevertheless, it was necessary to check this possibility experimentally. This was done in a study by R. T. Kazakova (Tyurina) and N. Yu. Leont'yeva [1971].

The experiments were conducted on four mongrel adult dogs. Unfortunately, the limited size of the chamber made it necessary to experiment on small dogs weighing from 7 to 10 kg. The animals were of average fatness.

In the first series of experiments, the dogs were raised in the chamber (filled with pure oxygen) to altitudes of 10, 11, 12 and 13 km and kept there for 2-2.5 h. In spite of repeating these experiments on each of the four dogs, in none of them did we succeed in producing any clear symptoms of the bends type.

At the same time, in a number of experiments, in particular at an altitude of 10 km, too, where the hypoxemic factor should not have acted, the dogs showed panting, which clearly attested to development of aeroembolic phenomena.

In the second series of experiments, the dogs were first subjected to supplementary saturation with nitrogen. To this end, they were first kept for different periods ranging from 2.5 to 10 hours (most often, five hours), in a compression chamber under pressures ranging from 0.5 to 2.8 gage atmospheres, and then transferred to a low-pressure chamber and raised to an altitude ranging from 8 to 11 km, where they were kept for another two hours.

Just as in the experiments without supplementary saturation, with supplementary saturation of the dogs with nitrogen under pressures of 0.5 and 0.8 atm and a subsequent two-hour stay at altitudes of 10,000-12,000 m, the authors could detect no clear and unmistakeable bends symptoms. In a number of experiments, though, the animals displayed anxiety, which could presumably be connected with painful sensations. The dogs began to hop about, sometimes raising one or another paw for a short time. It could be imagined that the dogs were feeling mild painful sensations that did not reach that intensity at which they lead to typical, outwardly noticeable malfunctioning of the extremities. At the same time, pronounced panting and general depression were noted, which were undoubtedly due to general aeroembolic phenomena. У308

In the next experiments, high values of increased pressure were used for supplementary supersaturation.

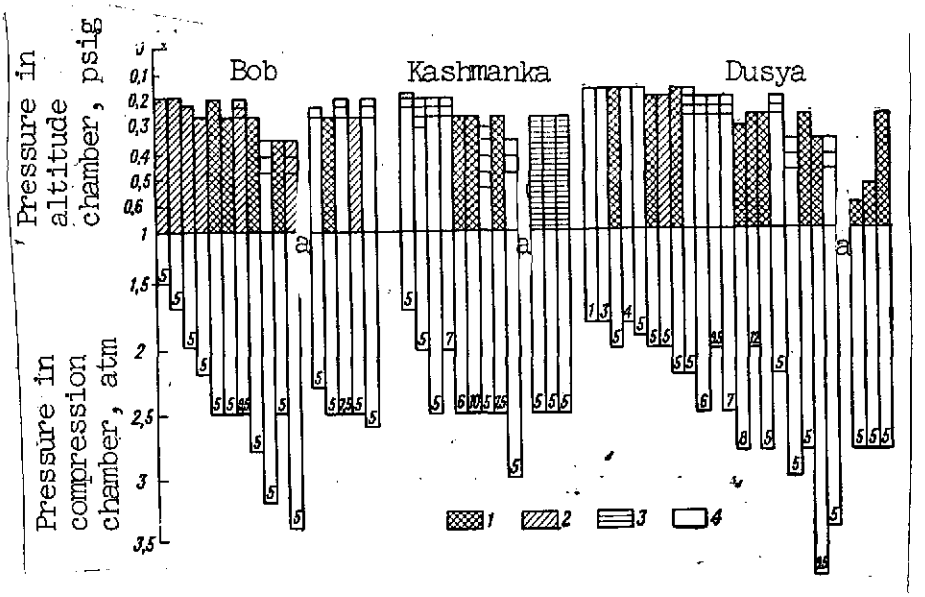

Fig. 75. Results of experiments with supplementary saturation of dogs with nitrogen in compression chamber and subsequent ascents to an altitude. Figures at the bottom of the columns are the dogs' staying times under increased pressure, h; 1 -- pronounced bends symptoms; 2 -- unclear signs of the bends; 3 -- acute deterioration of the dogs' general condition; ♯ -- absence of functional disorders.

Key: A. Interruption

The results of these experiments on three dogs (the fourth was used only in experiments without supplementary saturation) are shown in Fig. 75. The figure shows that clear symptoms of the bends were obtained in the dogs Bob and Kashmanka only after supplementary saturation with nitrogen under a pressure of 2.5 atm (1.5 gage atmospheres). In the dog Dusya they were first recorded after using a pressure of 1.8 atm (0.8 psig), and in subsequent experiments, after pressures of 2 atm and greater. The bends symptoms in the experiments in question were sometimes transient, but sometimes marked by considerable persistence and intensity; in these cases, the dogs hopped on three paws, drawing in the affected extremity, and whimpered and displayed motor unrest. Noteworthy are two experiments on the dog Dusya; when, /309 upon keeping the dog at the altitude, this symptom of the bends, which had appeared in one of the rear extremities, changed over to paresis and paralysis of the given extremity, it was joined by paresis of the second rear extremity and of the entire rear part of the body. After rapid descent to the ground, these

threatening manifestations did not disappear at once. It is very probable that they were mainly caused by the "depth" component of the experiment, i.e., preliminary supplementary saturation of the organism with nitrogen under increased pressure conditions; but their development under altitude conditions must, of course, come in for special notice.

The data of Fig. 75 show that the results of the experiments under discussion are not distinguished by constancy. Under identical conditions, bends symptoms sometimes arise, sometimes do not appear and are absent even from experiments involving still more drastic conditions. At the same time, these conditions are such that they undoubtedly lead to very considerable degrees of supersaturation of the organism with nitrogen.

The increased pressure values used incident to supplementary saturation approximate those at which decompression disorders already develop in a number of dogs incident to return to ordinary normal pressure without using any supplementary decompression (ascent to an altitude).

Somewhat puzzling and worthy of investigation is the fact that the dogs' resistance to the decompression disturbances that arise after their emergence from under increased pressure generally exceed only but little the resistance of human beings; but as soon as the atmospheric rarefaction factor is introduced into the matter, the differences in resistance become, so to speak, incommensurate.

Indeed, the values of the safe supersaturation coefficient upon emergence from under increased pressure in some of our dogs, especially in the initial experiments, were close, and in individual animals even equal, to the values of the SSC for human beings and corresponded to depths of 20, 18, 16 and even 14 m. At the same time, in the just-described experiments, the dogs in a number of cases comfortably endured such conditions of composite decompression from under increased pressure to a rarefied atmosphere as would probably be fatal for man. The results of our study using increased pressure with dogs of the most different sizes give no ground for explaining this circumstance by the small size and weight of the dogs employed in the experiments of R. T. Kazakova and N. Yu. Leont'yeva. Clearly. the explanation of this fact -- the sharp divergence in the resistance of dogs and people to altitude decompression disturbances -- is concealed in some internal peculiarities and intimate regularities of development of these disturbances in a rarefied atmosphere.

Thus, the experiments showed that bends symptoms in dogs /310
in a rarefied atmosphere are difficult to induce; they arise, and then far from in all cases, only under the condition of

preliminary supersaturation of the animals with nitrogen, the degree of supersaturation of the organism, if it is measured, as is customary, by the supersaturation coefficient, proving to be extremely high. We can have confidence that the muscular work of animals under conditions of rarefaction, on the analogy of the results of investigations on people, will to a great extent promote development of decompression symptoms of the bends type. The organization of such experiments with dogs running on treadmills under conditions of rarefaction constitutes one of the urgent tasks of future investigations.

We must say a few words about the attempts of R. T. Kazakova and K S. Yurova to induce development of altitude symptoms of the bends in dogs by applying a tourniquet to one of the rear extremities before the animal's ascent to the altitude for the purpose of restricting the blood flow in the given extremity and hindering the desaturation of its tissues from nitrogen. Individual test experiments gave some hope that such results could be obtained; but it became clear that before any more such experiments could be carried out, it would be necessary to work out a special technique of tourniqueting the extremity which would preclude ischemia and ensure curtailment of the blood flow.

Experiments carried out on rabbits by R. T. Kazakova and N. Yu. Leont'yeva for the purpose of inducing altitude decompression disorders with some clear criteria for them, yielded even less reassuring results than the experiments on dogs. A number of rabbits, especially fat ones, incident to ascents to altitudes of 11-12 km in an oxygen atmosphere, developed panting, depression and physical weakness, which are obviously the result of aeroembolic phenomena. Incident to ascents to an altitude (from 7 to 10 km) after preliminary supersaturation with nitrogen, the same phenomena set in. If the degree of supersaturation is increased, the above symptoms progress rapidly, and the rabbits die. Knowing from experiments with increased pressure that rabbits generally do not display clear signs of appearance (beginning) of decompression disorders, we did not increase the number of these experiments. It should be stressed, however, that in the experiments conducted on rabbits, that fact which emerged strikingly in experiments on dogs received confirmation, namely: introduction of the rarefaction factor into the experiments increases those differences in resistance to decompression disturbances which are noted between man and said animals incident to decompression from under increased pressures.

V. P. Katuntsev carried out experiments directed at inducing altitude decompression disorders in rats. Taking into account the very great resistance of rats to decompression disturbances incident to decompression from under decreased pressures, we /311
considered it useless to check only the effect of the rarefaction

factor in these animals and immediately introduced a physical load into these experiments. The rats were trained according to a method used by O. Yu. Sidorov and V. K. Sulimo-Samoyllo to run on a turning treadmill -- a wheel -- placed inside the altitude chamber. Incident to ascents to 10-12 km using a chamber filled with oxygen, V. P. Katuntsev has not yet succeeded in inducing clear signs of decompression disorders in rats running on a treadmill, although O. Yu. Sidorov and V. K. Sulimo noted such disorders in the form of a weakening of the animals and their giving up running on the treadmill, which, according to the data of the authors, was detected especially clearly when artificially induced hypercapnia was combined with altitude decompression. Using preliminary supersaturation of the rats with nitrogen incident to increased pressure, V. P. Katuntsev chose such conditions under which the rats at the altitude displayed decompression disorders that were sometimes manifested not only in the above-mentioned general manifestations but also in the preferential malfunctioning of one or two extremities. The impression was created that such experiments are not lacking in promise and can serve for study of additional effects that are capable of causing considerable changes in the organism's resistance to decompression disturbances.

Study of formation of gas bubbles in the blood of animals incident to the action of different degrees of atmospheric rarefaction

In early investigations, in addition to detailed analysis of the aeroembolic phenomena that develop in animals after decompression from under increased pressure, I undertook heuristic experiments involving study of gas formation in the blood in a rarefied atmosphere.

On six cats and two dogs under anesthesia, we conducted crucial experiments involving rapid ascent of the animals to an altitude of 8 km (two experiments on cats) and 10 and 12 km (two experiments on cats, one on dogs). From the start of ascent, the animals were switched over to oxygen breathing. To check for gas formation in the blood, we used gas traps introduced into the femoral veins. Incident to ascents to altitudes of 8 and 10 km, not a single case of gas bubbles in the venous blood was detected. Incident to ascents to an altitude of 12 km in two cases (in a dog and a cat), 7-8 min after termination of rarefaction single gas bubbles were detected in the gas traps.

Besides these experiments, on three cats and four rabbits we conducted crucial experiments involving inspection for gas-bubble formation in the mesenteric vessels of the intestine drawn out from the abdominal cavity and stretched out fanwise incident to ascent to altitudes of 10, 12 and 13 km. At an /312

altitude of 10 km, gas bubbles were absent from the mesenteric vessels; at altitudes of 12-13 km, in three out of five cases the appearance of single gas bubbles was noted in the mesenteric veins.

These facts confirmed the possibility first suggested by Armstrong that bubbles might appear in the blood incident to those degrees of rarefaction which man may encounter under special conditions of activity. But the experiments referred to were not designed to elucidate the probability of intravascular gas formation incident to different degrees of rarefaction or to determine the minimum altitudes at which gas bubbles can appear in the blood. At present, elucidation of these questions has become necessary.

It became clear from investigations of people that incident to the rather prolonged action of a rarefied atmosphere on a man performing muscular work, decompression disorders can arise throughout the range of pressures that are used in spacesuits, i.e., at altitudes ranging from 7-11 km. R. T. Kazakova, who has been especially occupied with the question of decompression disorder development at low altitudes, recorded a case of clear bends symptoms arising at an altitude of 6 km, as well as the above-mentioned case of mild painful sensations appearing in one of the examinees at an altitude of 5 km. An analysis of the literature that she undertook showed that the facts of decompression disorder development at such altitudes, as single cases, have been noted by other researchers, too. In connection with this, it was natural to set ourselves the task of elucidating the possibility of gas formation in the organism, especially in the blood, at different, and especially minimal, altitudes. Jointly with N. Yu. Leont'yeva, R. T. Kazakova undertook crucial experiments on cats using the earlier glass gas traps and the effect on animals of different degrees of atmospheric rarefaction. Barbiturate narcosis was employed, the animals were massively heparinized and gas traps were inserted in the rear vena cava. The experiments were carried out in two versions -- with ascents of the animals immediately to one or another definite altitude and exposure there for 30-40 min and with stepped transition from lower to higher altitudes with an initial exposure of 30 min and subsequent exposures -- at each kilometer -- of 10 min. The experiments in which bubbles did not appear at altitudes less than 12-13 km were topped off with ascents to these extreme altitudes.

The results of the experiments are shown in Table 19.

They show that under these experimental conditions, the boundary of rather probable appearance of single gas bubbles that are visible to the naked eye in the venous blood falls in the

TABLE 19. RESULTS OF EXPERIMENTS INVOLVING STUDY OF GAS FORMATION IN THE VENOUS BLOOD OF CATS AT DIFFERENT ALTITUDES

Altitude, m	Total number of experiments	Number of cases in which bubbles were found	Percent of total number
3 000	30	1	3
4 000	36	8	22
5 000	38	16	42
6 000	39	18	46
7 000	34	17	50
8 000	35	22	63
9 000	25	18	72
12 000	18	15	83
13 000	46	44	96

4-km altitude range. The general regularity reflecting the frequency (probability) of appearance of gas bubbles in the blood at different altitudes is best brought out in the figures of the right-hand column of the table, where for each altitude /313 the number of cases of bubbles detected is expressed in percents of the total number of observations. (We have taken the liberty of deviating from the rule according to which percents are by definition determined from starting figures that are not less than 100 in view of the fact that the indicated regularity is sufficiently clear and definite). In Fig. 76 it is depicted graphically. It can be seen from the curve that a real, rather high probability of appearance of visible gas bubbles in the blood (under these experimental conditions) sets in at an altitude of about 4 km; toward 5 km, it increases sharply, and then begins to increase more slowly. In view of the small number of observations, there are scarcely any grounds for crediting the inflections of the curve in the range from 5 to 13 km. It must be supposed that in this altitude range, the probability of appearance of gas bubbles in the blood increases uniformly in direct proportion to the degree of rarefaction of the atmosphere. This is suggested by simple logic, as well as by analogy of the probability of development of bends symptoms in human beings at altitudes from 7 to 12-13 km (on this assumption, it is necessary to take into account that between appearance of bubbles in the organism and development of visible decompression disorders, as was shown above, there is always a certain gap). Be that as it may, the presented curve shows that as applied to the given experimental conditions, the real probability of formation of

visible gas bubbles in the blood sets in only at certain altitudes; at an altitude of 7 km it amounts to 50%, while at an altitude of 13 km it approaches 100%. On the other hand, what is no less significant is that it can be seen from the shape of the left part of the curve that the probability of gas formation, if we go from 5 km downwards, drops steeply and toward an altitude of 3 km, it practically dwindles to nothing. Examination of the presented regularity creates the distinct impression that there exists an altitude range that is safe in respect of formation of gas bubbles that are visible to the naked eye.

It should be recalled that above, it was a question of the /314
qualitative side of the matter, i.e., the fact of appearance of gas bubbles; if we take up its quantitative side -- the number of bubbles accumulating in the gas trap --, then we find the following. At altitudes of 4-5 km, single or even solitary gas bubbles generally appear in the gas trap. But in the altitude range from 6-7 to 12-13 km, the number of bubbles, as a rule, proves to be more considerable and generally increases in proportion to the altitude. But very often individual differences in the animals get the upper hand of this general regularity. In some, abundant gas formation can take place at altitudes of 8-9 km; in others, even at extreme altitudes (12-13 km), single gas bubbles appear in the gas trap. Our attention is also attracted by those cases, not numerous though they are in the present series of experiments, where even at extreme degrees of rarefaction, no gas bubbles were detected in the blood. Upon autopsy of the animals at the end of the experiments, careful inspection of the different vessels and cavities of the heart disclosed the same facts.

It is necessary to dwell on still another question that is of importance as a matter of principle not only for interpretation of the results of the experiments with gas traps on which we have reported above, but also for elucidation of the conditions of postdecompression gas formation in the intact organism -- in the venous blood of different animals and man. It will be a question, on the one hand, of the time required for formation of gas bubbles that are visible to the naked eye in the supersaturated blood and, on the other, of its flow rate. I have already touched upon these circumstances earlier in discussing the possibility of gas formation in the arterial blood after decompression from under high pressures. Now it is necessary to evaluate their significance for the process of formation of gas bubbles in the venous blood.

Let us for a moment digress from the results of the experi- /315
ments with gas traps and examine this question as applied to the intact organism and conditions of ascent to an altitude. In order to elucidate this question, it is necessary to imagine what consequences decompression leads to from the very start. From the start of decompression and at the altitude, when around the

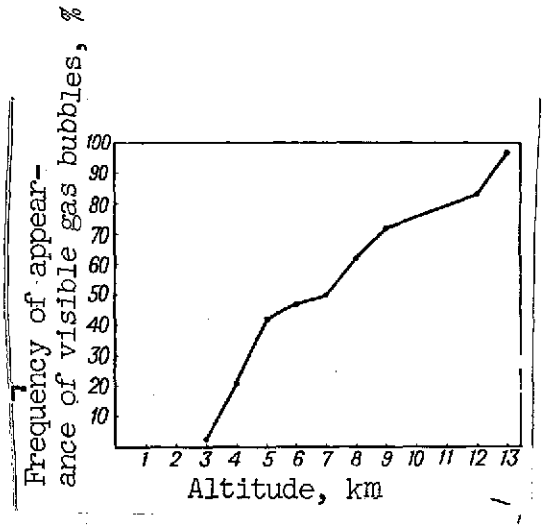

Fig. 76. Probability of appearance of visible gas bubbles in the venous blood of anesthetized cats at different altitudes in crucial experiments using gas traps (according to data of R. T. Kazakova and N. Yu. Leont'yeva.

organism and in its lungs an oxygen medium with an increased pressure is created, the oxygen contained in the blood on passing through the pulmonary capillaries, diffuses from the alveoli. If we consider, as it is customary to do, that the blood while flowing along the pulmonary capillaries gets into complete gaseous equilibrium with the alveolar air not only for oxygen and carbon dioxide, but also for nitrogen, then we must suppose that soon after transition to oxygen breathing, the arterial blood already proves to be constantly desaturated from nitrogen. Passing along the capillaries of the tissues, it takes up the nitrogen diffusing from them, and if we again proceed from the fact that the gaseous equilibrium between blood and tissues is established rapidly and completely, this means that the blood flowing from the tissues will prove to be supersaturated to the same extent as the tissues themselves. At a certain degree of supersaturation, gas bubbles can be formed in the blood. But it takes a certain time for gas bubbles that are visible to the naked eye to be formed. The greater the degree of supersaturation, the shorter this time and, conversely, the lesser the supersaturation, the longer this time. Here it is that the question asked above arises: how great is the supersaturation of the venous blood and how rapidly does it make its way from the sources of the venous system, where its maximum supersaturation was obtained, to the pulmonary capillaries, where the nitrogen begins to pass rapidly out of the blood, and there is no gas formation?

The possibility that gas bubbles that are visible to the naked eye may be formed in the venous blood is directly and immediately determined by these two temporal parameters: the time it takes for bubbles of the size in question to be formed incident to the given degree of supersaturation of the blood, and the time it takes the blood from the capillaries of the greater circulatory system of tissues of one kind or another to reach the pulmonary capillaries. It is quite clear that this kind of passage of the blood from the tissue capillaries to the pulmonary capillaries in the organism of each individual animal and each

individual human being differs sharply for different regions of the body -- more exactly, for individual regional circulatory systems that differ in length. It is shortest for the coronary circulatory system and longest for the circulatory systems belonging to the distal parts of the body that are most remote from the heart. True, this circumstance -- the remoteness of tissues of one kind or another from the heart -- does not yet completely determine the time of passage of blood from the tissues to the pulmonary capillaries. Thus, for example, the brain of all animals and man is located at a rather considerable distance from the heart; but the circulation of the blood through the system of the cerebral vessels takes place relatively rapidly, taking less time than in some other tissues that are less active in /316
their function (for example, adipose tissues) but situated at the same distance from the heart. In other words, the time of passage of the blood from the tissues to the pulmonary capillaries is determined not only by the distance of the corresponding regions of the body from the heart, but also by the state of the vessels and the intensity of the regional blood flow in the corresponding regions. Be that as it may, if we take an individual organism, then among the countless multitude of individual regional parallel circulatory systems belonging to individual organs, regions of the body and tissues, there are such in which the time of passage of the blood from the tissue capillaries to the pulmonary capillaries turns out to be greater. In the venous blood passing along these circulatory systems, ceteris paribus, the probability that gas bubbles that are visible to the naked eye may be formed incident to decompression is greater. And though these bubbles can already be detected in the total venous blood (in the main veins, the right side of the heart), they must clearly be attributed to those flows of venous blood which take the longest time to make their way from the tissues to the main vessels. During this time, the gas bubbles pass through the initial stages of their formation and growth. But let us return from these details concerning each individual organism to the specific differences in the general intensity of blood circulation in different animals. With these great differences it is legitimate to connect the very sharp difference in the resistance of animals of different species to decompression disturbances. But in speaking of this, what is usually had in mind is only one circumstance, namely, the rapidity of desaturation of the tissues and of the organism as a whole from the inert gas. The significance of this circumstance is clear and incontestible. But, as ensues from all of the foregoing, together with it there emerges another circumstance connected with the general intensity of the blood flow -- the rate of passage of the blood from the tissues to the pulmonary capillaries -- which determines the possibility of development of aeroembolic phenomena in the venous blood. Of this it is easy to convince oneself by subjecting different animals (for example, dog, cat, rabbit, rat and mouse) to deliberately complete saturation with nitrogen incident to an

air pressure in the chamber of about 4 atm and subsequent single-step decompression down to normal pressure. As a result of this kind of experiment, we shall see sharp functional disorders in the dog and rabbit and, most likely of all, we shall witness the death of these animals. We shall find more or less serious functional disturbances in the cat, which attests to development of general aeroembolic phenomena, though the cat will in all likelihood survive these disorders. And we shall not note any sharp functional shifts in the rat or mouse.

At the same time, if we visualize the picture of saturation of the blood with nitrogen in these animals in the earliest periods after decompression, we shall be obliged to acknowledge /317 that in this picture, there are no differences of principle between any of these animals. In all of them, the arterial blood that has passed through the lungs, where the partial nitrogen pressure was close to the ordinary, will no longer be supersaturated with this gas, and in all of them, the venous blood will be characterized by the same supersaturation inasmuch as all tissues in the organism of all of the animals were rather fully saturated with nitrogen under identical conditions. Consequently, the only circumstance that determines such a difference in outcome of this experiment for different animals is the great difference in the time of passage of the venous blood supersaturated with nitrogen from the tissue capillaries to the pulmonary capillaries. In the rabbit and dog, this time will suffice for mass gas formation that is incompatible with life to take place in the venous blood; in the cat, this time will be shorter, and aeroembolic phenomena will develop in a pronounced, though most likely not fatal, form; finally, in the rat and, especially, the mouse, this time will be so short that gas bubbles of pathogenic sizes will not succeed in being formed to any considerable extent. If we turn to the conditions of ascent to an altitude without desaturation, what first of all emerges here is the fact that the degrees of supersaturation of the organism as a whole, of its tissues and, consequently, of the venous blood, too, will be less than in the example under consideration. Incident to altitude decompression for those cases which are of especial interest to space practice (changing over to a spacesuit), this degree of supersaturation will always be limited by the range of possible drops in pressure (at most, from 120.22 atm) and can never attain the values that are encountered in diving practice. At lesser degrees of supersaturation, as has already been pointed out, the time needed for bubbles that are visible to the naked eye to be formed, will be greater. Incident to ascents not to maximum permissible (according to the conditions of oxygen provision) altitudes (10-11-12 km), but to lesser altitudes (9-8 km, etc), it will increase still more, in conformity with which the probability of formation of visible gas bubbles in the venous blood will decrease.

Returning now to the experiments of R. M. Kazakova and N. Yu. Leont'yeva with gas traps that were presented above, we must

evaluate their results with due regard for the circumstance that the blood flow slows down in gas traps, that gas traps increase the time of passage of the blood from the tissues to the lungs and thereby promotes appearance of gas bubbles in the blood. True, the volume of the gas traps used by the authors was small and fell in the range from 5 to 6 mℓ, but in spite of their comparatively small volume, the slowing down of the blood flow cannot, of course, be left out of account. Hence it would be rash to transfer the results of these experiments to the intact organism of the same animals and to assume that the process of incipient formation of visible gas bubbles in the venous blood /318 corresponds to the same altitude limits that emerged in these experiments. But in our opinion, this circumstance does not deprive the indicated experiments of considerable interest and significance. Their results can, in our opinion, be regarded as rather direct evidence showing at what altitudes those degrees of supersaturation of the venous blood with nitrogen (more accurately, the total partial pressures of all of the gases dissolved in it) are attained at which it becomes possible for gas bubbles that are visible to the naked eye to appear in the blood. It should be noted that some adherents of Harvey's theory about the role of "gas seeds" in forming postdecompression gas bubbles are inclined to be skeptical with regard to the results of the experiments with gas traps on the ground that the operation of introducing them into vessels, as well as these instruments themselves, cause the indicated "gas seeds" to be introduced into the blood, which is what leads to the process of gas formation in the supersaturated blood, which, according to these critical judgments, might not have taken place if gas traps had not been used.

Here I shall not enter into a lengthy polemic with the authors of such critical remarks or in discussion of a subject that is not amenable either to objective recording or to observations -- "gas seeds" --, but shall confine myself only to referring to the obvious facts that were stated earlier, facts which permitted experiments using gas traps incident to study of the aeroembolic phenomena that arise after decompression from under increased pressures. These experiments showed that what we can see in gas traps can be observed under the same experimental conditions in intact prepared vessels, too, it being a question not of excessive degrees of supersaturation of the organism and blood nor of mass gas formation in it, but of threshold supersaturations and incipient appearance of gas bubbles. Thus, as a rule, incident to appearance of gas bubbles in a gas trap inserted in the femoral vein of a dog, we can see them appearing in the venous vessels of the contralateral extremity or in the vessels of the subcutaneous cellular tissue of different regions of the body. Researchers who are familiar with vivisection know how easily and bloodlessly (according to the expression of surgeons, "in a dull way") the skin with its subcutaneous cellular

tissue can be separated from the relevant tissues in such a manner that the subcutaneous vascular network and the vessels of the relevant tissues thus revealed for observation will remain uninjured and the entry of "gas seeds" into them will not be very probable. Under the same conditions, we can see gas bubbles also in other mesenteric vessels looking over the intestine throughout its length, when there can be no question of any rupture of these tissues.

The direct and clear correlation between the amount of gas bubbles that can be recorded with the aid of gas traps inserted in one of the femoral veins of the dog and the functional shifts /319 involving the respiration and blood circulation also compel us to reject the idea that the gas trap in itself determines development of aeroembolic phenomena in the organism. Here, by the way, we must take into account the fact that the gas trap delays gas bubbles, and the indicated functional shifts are caused not by these delayed bubbles, but by others coming from uninjured regions.

Nor, finally, does the assumption that any vascular trauma (introducing a cannula for measuring blood pressure, or just cutting any peripheral tissues) can, because of "gas seeds" introduced into the blood, be the determining cause of development of postdecompression aeroembolism, bear experimental checking. Of this any one can convince himself by comparing the consequences of decompression from under different pressures in paired experiments on two animals of the same species that are identical in all of their data, first anesthetizing these animals, and then, before or after decompression, subjecting one of them to vivisectional effects of one sort or another -- of course, not too serious ones. Neither in development or degree of evidence of symptoms of postdecompression aeroembolism, nor incident to intensification of the effects, will any difference in the outcome of the experiments be obtained as between the untouched animal and the animal traumatized to one (not excessive) extent or another.

Thus, all of the adduced facts and all of our experience in using gas traps incident to recompression from under increased pressure, as well as incident to ascents to an altitude, created in us the impression that the possible accumulation of any microscopic "gas seeds" in the vascular system is not a decisive matter of principle for the results of the experiments. The possibility of microscopic "gas seeds" being introduced into the blood incident to inserting gas traps cannot, of course, be completely denied; but all of the circumstances examined above argue in favor of the fact that if this phenomenon does take place, it is not determinative for appearance of gas bubbles either in gas traps or in other sections of the vascular system.

On the basis of everything that has been said about the results of the experiments of R. T. Kazakova and N. Yu. Leont'yeva, we can conclude that the disclosure of gas bubbles at the indicated altitudes reflects that degree of supersaturation of the blood with nitrogen in summation with other gases at which the given process begins (at altitudes of about 4,000 m) and develops. But the circumstances are favorable for the organism in this respect that, as a rule, incident to unchanged blood flow, the time of passage of the blood from the tissue capillaries to the pulmonary capillaries proves to be insufficient for gas bubbles to be formed and to grow to sizes that are visible to the naked eye.

Confirmation of the foregoing can be found in the results of heuristic experiments carried out by R. T. Kazakova and V. P. /320
Katuntsev on anesthetized cats and rabbits, in which ultrasonic pickups were placed on their inferior vena cava and the passage of bubbles along the vessels was recorded according to the principle of the Doppler effect. In 14 experiments on cats involving ascent to an altitude of 12,000 m (with oxygen provision), not a single case of gas bubbles was detected by the indicated method. When one cat was raised to an altitude of 14,000 m, signals attesting to gas bubbles passing along the vein were recorded. Out of 16 experiments on rabbits at an altitude of 12,000 m, gas bubbles were recorded in half of the cases. The effects were sometimes manifested in single signals pointingtto passage of gas bubbles, but sometimes attested to their mass appearance.

Thus, between those altitudes at which supersaturation of the venous blood is attained, incident to which gas bubbles that are visible to the naked eye can be formed, and those altitudes at which this possibility can be realized in the intact organism, there exists a considerable gap. It is very probable that this gap can be decreased as a result of intensified production of carbon dioxide in the tissues and an increase in its pressure in the venous blood. This circumstance, which has been analyzed in detail by Whitaker and his collaborators, must undoubtedly be of rather great significance for development of altitude decompression aeroembolism.

It should be noted that a conclusion of importance from the practical point of view ensues from the above data of R. T. Kazakova and N. Yu. Leont'yeva on supersaturation of the venous blood that is sufficient for gas formation already at comparatively low altitudes. It is a question of possible instances of slowing down of the rate of blood flow, for example in the extremities, for mechanical reasons (pressing of the tissues of the extremities by the clothing worn under the spacesuit, forced unnatural position of the extremities). It is likely that the genesis of gas bubbles in the corresponding vessels under such circumstances may stimulate gas formation also in the general flow of the venous blood in the main vessels.

To everything that has been said about the process of gas formation in the venous blood must be added that this process is to a considerable extent subject to the influence of some internal individual factors concerning apparently the physico-biochemical properties of the blood. This circumstance already emerged rather clearly in the experiments with gas traps, when under identical conditions, in some animals single gas bubbles were noted, while in others they appeared in mass amounts. This fact was confirmed not only by observing gas traps, but also by opening animals after experiments, incident to inspection of vessels of different regions. In order to check this fact some more, R. T. Kazakova and K. S. Yurova undertook experiments in which rats were killed under ordinary circumstances, /321 their corpses were raised to an altitude of 12 km with a 2-hour exposure there and were subsequently lowered to the ground and cut open with careful inspection of the vessels of different regions. These vessels also showed that the intensity of gas formation in the blood of killed animals varies greatly, and in a number of cases, no gas bubbles at all can be detected in the vessels. This fact is undoubtedly worthy of serious attention. So far, K. S. Yurova and N. Yu. Leont'yeva have undertaken only the first steps in its analysis. A statistically significant dependence was detected between the initial quantity of leukocytes in the blood of the animals and the intensity of gas formation in subsequent experiments with their destruction. As a rule, the greater the quantity of leukocytes, the greater the intensity of gas formation in the blood. Also statistically significant proved to be the dependence of intensive gas formation on the age (weight) of the experimental animals. With increasing age (weight), the probability of mass gas formation under the indicated experimental conditions increases.

As has already been pointed out, one of the most important questions from the theoretical, as well as practical, point of view in the study of decompression disorders is the following: under what conditions and at what degrees of supersaturation does the process of gas formation in the organism (in the blood, as well as in the tissues) begin? It has not yet been cleared up whether this process is determined by certain critical degrees of supersaturation (coefficient of maximum permissible supersaturation) or whether fine gas bubbles can begin to be formed from the very start of decompression. Solution of this question is very complicated because the beginning of the process of gas-bubble formation in the organism's different media is hidden from the observer's eyes and is not yet amenable to objective determination. The experiments conducted by R. P. Kazakova and K. S. Yurova with the participation in some of them of V. P. Nikolayev that have been discussed in the present section are an attempt to approach solution of this question.

As a method of procedure for study of this question we decided to make use of the fact that the buoyancy of the animals'

corpses changes when gas bubbles are formed in them. The bubbles that are being formed occupy a certain volume in the corpse, in virtue of which the total volume of the corpse increases as the bubbles are formed. The absolute weight of the corpse does not change, but its specific weight decreases and its buoyancy increases.[18]

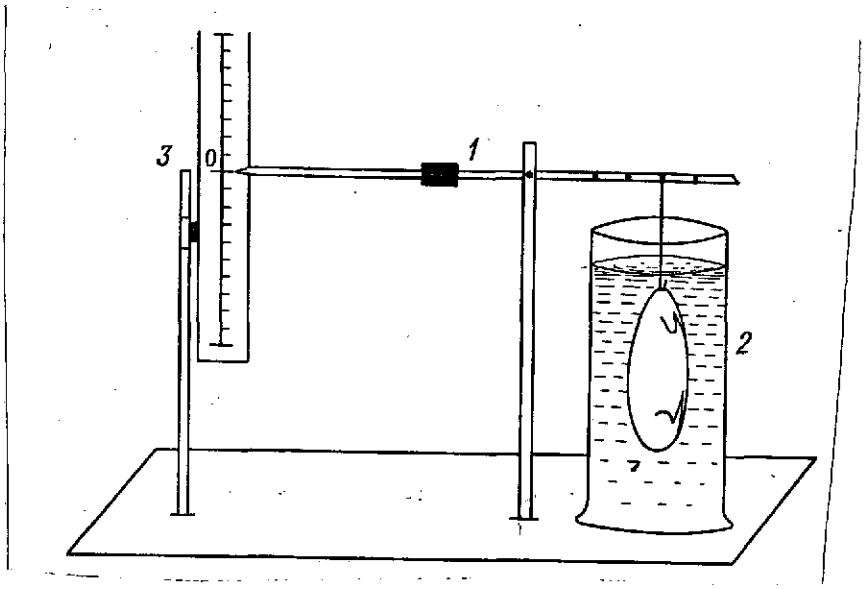

Fig. 77. Diagram showing setup for determining the change in buoyancy of a rat's corpse (carcass) under the action of a rarefied atmosphere. 1 -- lever with cursor; 2 -- carcass of rat in vessel with water desaturated from gases; 3 -- scale for recording changes in buoyancy.

The idea behind the experiments was to determine the changes /322 in buoyancy of a rat's corpse (carcass) at different altitudes and to ascertain whether these changes arise only at a certain degree of rarefaction or whether they take place from the very beginning of decompression, i.e., even at the lowest altitudes.

The rat specimen was prepared in the following manner: the

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[18] Soon after beginning our experiments based on the given method of procedure, we learned from an article recently published by Behnke [1971] that the fact of a significant decrease in specific weight (increase in buoyancy) of fat dogs killed after a stay under increased pressure has been noted in experimental practice.

rat was killed and carefully depilated. Its head and tail were cut off, and all of its cavitary organs were removed through a wide ventral cut. The thusly prepared carcass was suspended from one arm of a two-armed lever and immersed in a cylinder of well-boiled water. As can be seen from Fig. 77, the lever with the carcass was counterbalanced with a runner travelling along the lever's free arm. The pointer of the lever was set at the zero point of a vertically attached scale. The entire system with the specimen was placed in an altitude chamber and subjected to the action of a rarefied atmosphere.

Carried out so far have been for the most part only preliminary experiments (42 of them) aimed at working out and standardizing the method, and 18 test experiments were set up with atmospheric rarefactions corresponding to an altitude of 10,000 m.

Without entering into the details of the obtained results, the latter can be summarized as follows. Incident to ascents to an altitude of 10,000 m, the buoyancy of the rat's carcass /323 increases rapidly in the very course of decompression. This effect is evidently connected with an increase in volume of previous gas bubbles located in some places of the specimen. These may have been small recesses in the folds of the surface of the carcass or in the upper parts of the ravaged thorax. Be that as it may, this effect of rapid increase in buoyancy of the carcass incident to ascent is indubitably not connected with the onset of new gas bubbles and is therefore of no great interest in itself. What attracts the attention is another fact: a gradual and steady increase in buoyancy during exposure at the altitude after termination of ascent. This fact can in no way be connected with expansion of previous gases but can be nicely explained by formation of new gas bubbles at the altitude. The increase in buoyancy usually continues for 70-90 min, after which the curve changes into a plateau. Incident to descent, buoyancy decreases, for the most part without returning to its initial level. If after this the animal's corpse is again raised to an altitude, the effect of rapid increase in buoyancy incident to this ascent is considerably greater than incident to the first. This fact indicates that the amount of free gas in the corpse increases after prolonged exposure at an altitude. The described effects are illustrated by Fig. 78, which reflects the results of one of the experiments. Significant are the results of individual test experiments with supersaturation of a rat (until its death) with nitrogen under 0.5 psig overpressure and with preliminary desaturation of the animal. In the first case, the increased buoyancy effect at the altitude is considerably intensified, while in the second case, it is sharply weakened.

These facts argue in favor of gas-bubble formation in the /324 animal's corpse in a rarefied atmosphere. True, one doubt

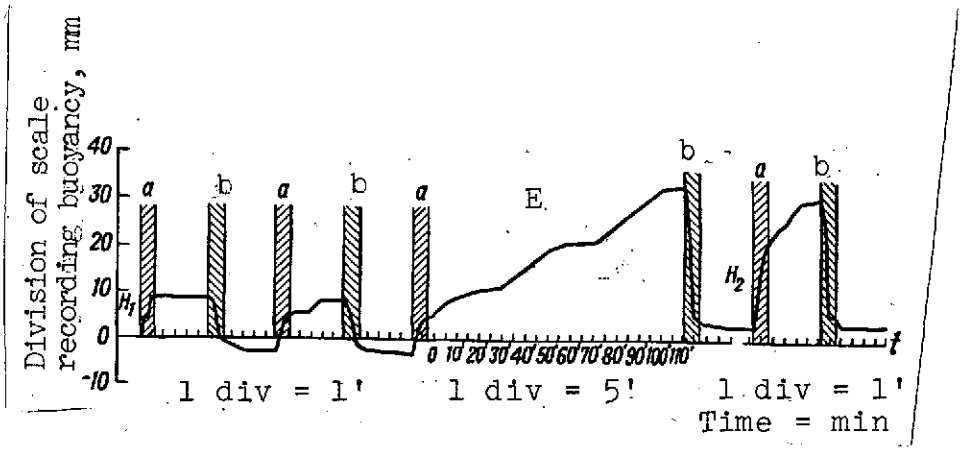

Fig. 78. Changes in buoyancy of corpse (carcass) of rat incident to ascents to 10,000 m. H_1 -- increase in buoyancy incident to first ascent; H_2 -- increase in buoyancy incident to final ascents; E -- changed buoyancy effect incident to 2-hour exposure of specimen at 10,000 m; A -- ascents; B -- descents.

remains: if gas bubbles or some gas accumulations preexisted in the corpse, incident to exposure at the altitude they may grow because of diffusion into them of nitrogen from surrounding tissues. But this hypothesis is by no means more probable than that of formation of new gas bubbles. It is possible that both are true. Be that as it may, the obtained facts require analysis and the original idea behind the experiments required execution: it is necessary to check whether this slow increase in buoyancy takes place only beginning with a certain altitude or whether it can be noted (of course, on a reduced scale) also at very low altitudes.

On the localization of gas bubbles that cause painful decompression symptoms

The question of the localization of the gas bubbles that cause symptoms of the bends is as old as the very history of scientific treatment of the problem of decompression disturbances. But owing to methodological difficulties, it has not been solved to this day and remains in the realm of speculation and discussion. Inevitably the researcher is confronted with this question every time he observes bends symptoms developing or feels them in himself and it obliges him to look over in his mind different hypotheses about the localization of pathogenically acting bubbles and to extract from his observations indirect arguments for the

correctness or incorrectness of these hypotheses. Of course, having so many times observed these decompression symptoms developing in dogs after decompression under ordinary conditions, as well as incident to ascents to an altitude, we could not but also ask ourselves where the gas bubbles that cause these functional disorders are located. In the present section, we shall discuss some facts that have a bearing on this question, and the results of experiments specially devoted to it.

Of the different hypotheses about the localization of the gas bubbles that cause bends symptoms, only one rests on certain experimental data. This is the hypothesis that the gas bubbles that cause painful sensations and functional disturbances in the extremities are formed from the synovial fluid and are located in joint cavities.

V. A. Skrypin [1948] has presented X-ray data on the presence of gas accumulations in the knee-joint cavity of man incident to development of painful symptoms in the region of this joint at an altitude of 9,000 m.

Thomas and Williams [1945] have reported that they managed to detect by means of X-rays gas bubbles in joints at an altitude of 7,000 m when painful symptoms were still absent from the examinees. /325

To check the hypothesis of the intrajoint localization of the gas bubbles that cause bends symptoms, on five dogs we undertook experiments in which the animals had air injected into their knee-joint cavity and were then raised to an altitude. In the initial experiments, we confined ourselves to injecting 1-2 mℓ air, while in subsequent experiments, we increased its dose to 5-7 and 10 mℓ. We took all measures to prevent the air from seeping back out of the joint through the wound entrance made by the needle. We used very fine needles; the puncture was made not in a single straight direction, but with different changes in the course of the needle as it penetrated into the tissue: piercing the skin, we pushed it aside together with the needle; pushing the needle forward there in the relevant tissues, we again changed its course and only then punctured the joint capsule. In a number of experiments, we shaved the site of the future puncture and, after injecting the air, at the same time that we withdrew the needle we covered the surface of the skin with a layer of adhesive or heated Mendeleyev's cement. There can be no doubt that the air always got into the joint cavity without fail. We checked ourselves by injecting dye in precisely the same manner into the knee-joint cavities in the corpses of the dogs used in the experiments, and incident to section, always convinced ourselves that the dye was actually inside the joint.

In none of the five dogs did an air injection into the knee-joint cavity, even in a dose of 5-7 mℓ, cause any sharp reactions or visible functional disturbances under ordinary pressure. When the dogs were raised to an altitude (up to 7.5 km and, in a number of cases, at an accelerated rate) immediately after the air injection, no symptoms similar to those of the bends arose in them. Only after injecting 10 mℓ air and more did it become noticeable that the dog was experiencing some discomfort in the use of the extremity; but neither on the ground nor at the altitude did these signs resemble even the incipient manifestations of decompression disturbances, not to speak of acute bends symptoms. They manifested themselves in the fact that the dog crossed its rear paws and sometimes looked around and stretched its snout out toward the extremity into whose joint the air had been injected, but none of these actions resembled the reactions elicited by pain; the impression was created that the animal was experiencing some unusual, but not painful sensations from the air injected in a large amount. Neither the initial lightening of the load on the extremity that is typical of decompression disturbances nor its subsequent flexions or, especially, the general reactions to manifest pain arose in the animals when such considerable amounts of air were injected into the joint.

Similar experiments were recently carried out by R. T. Kazakova on some of the dogs that she had used in the above-described experiments aimed at inducing altitude decompression disorders. She got the same results. No manifest signs of painful sensations reminiscent of bends symptoms were detected when the dogs were raised to an altitude after injecting air into their knee-joint cavities. Thus, the results of these experiments argue against the intrajoint localization of the gas bubbles that cause bends symptoms. /326

The use of the indicated method of procedure in the experiments of R. T. Kazakova was somewhat expanded. The author undertook air injections with all of the above-mentioned precautions not only in the knee-joint cavity, but also into the muscles and tendons of dogs and then observed their behavior incident to ascents to an altitude. Used most often for the air injection was the Achilles tendon (1.5-2 cm above the hock). Of course, the quantities of injected air were in these cases small, on the order of 0.3-0.4 mℓ. With this procedure, the dogs displayed some anxiety. But very soon after termination of the injections, no deviations from the norm could be observed in the dogs' behavior or condition or in the position of the extremity into whose tendon the air had been injected. Nor did subsequent ascents of the animals to an altitude bring out any signs that might attest to the onset of painful symptoms. It follows, as it were, from these experiments that neither the muscles nor the tendons (in any case, the Achilles tendon) are the seat of the gas bubbles that cause painful altitude symptoms to develop, either.

Arresting in connection with this question are experiments conducted on people by V. P. Katuntsev, who used mechanical pressure on the same regions in which the examinees felt altitude pains. The author used in her experiments a rubber pneumatic cup similar to the cup of a Riva-Rotchi apparatus. As soon as altitude pains arose, most often in one of the knee joints or in the immediately adjacent regions, the examinee, in accordance with a prearranged instruction, applied the cup to the given region. After the cup was applied, the experimentor gradually increased the pressure in it, the magnitude of which could be recorded. As the pressure was increased, the examinee reported on all changes in his sensations. The experiments yielded very clear and unambiguous results. In 32 out of 34 cases of using this procedure, incident to development of painful symptoms at different altitudes, when the pressure in the cup was increased, these symptoms weakened, and at a certain overpressure value, they disappeared in a preponderant number of experiments.

Significantly, in a great number of experiments the disappearance of the painful symptoms could be observed at that overpressure in the cup which, together with the surrounding /327
pressure at the altitude where the test was conducted, amounted to a pressure corresponding to altitudes of 5,000-6,000 m. It appears from all investigations on people involving development of the bends at higher altitudes that incident to descent, these symptoms disappear most often at a rarefaction corresponding to 5,000-6,000 m. The conclusion that ensues from these experiments by V. P. Katuntsev is, first of all, that the gas bubbles that cause painful symptoms are clearly not located inside the tubular bones. The hypothesis that they are localized here has been put forward by a number of authors. Some of them have supposed that the gas bubbles that arise in intraosseous vessels have no free egress through the narrow osseous apertures through which these vessels pass; others have considered it probable that the bends-inducing gas bubbles are formed in the osseous adipose tissue. Neither of these hypotheses agrees with the above results. The overpressure created from the outside with the aid of the cup cannot reach the intraosseous cavity or any of its contents. We are left with the thought that the gas bubbles that cause pain are located outside of the bones, in some media or tissues situated between the bone-supporting apparatus and the periphery.

Here again we can make two hypotheses: either that the gas bubbles are located within the vessels of the noted regions and cause pain because of ischemic phenomena or that they have an extravascular tissue location. The clear effects of using an overpressure cup are more in agreement with the latter hypothesis. These effects agree nicely with the conception of Nims [1951] that was mentioned earlier, according to which painful symptoms are determined by the magnitude of the overpressure in the gas bubble or, in other words, by a deformation pressure acting from within the bubble on the surrounding tissues. The counterpressure

applied from without must, to a certain extent, compensate for the overpressure in the bubble and decrease deformation effects.

A simpler question that has an immediate bearing on the question of where inside the organism, in which of its media or tissues, the gas bubbles that cause painful symptoms are localized is that of the localization and nature of the painful sensations themselves. This latter question, easy to study as it might appear, has not been subjected to sufficiently detailed systematic investigation, especially from the standpoint of comparing the localization and nature of painful altitude symptoms with painful bends symptoms after a stay under increased pressure. In the course of our investigations, V. P. Katuntsev conducted a very laborious and thorough study of this question. He set himself the special goal of systematizing all of our material concerning the localization and nature of altitude decompression pains. The second goal the author set himself was careful analysis of the data in the literature on the localization and nature of /328 the bends symptoms that arise incident to emergence from under increased pressure.

The general purpose of the entire study was comparison of these and other data. The results of analyzing our own material on the localization of altitude pains are reflected in Fig. 79. They show that the examinees in our experiments suffered mostly in their knee joints, and next in their talocalcaneal, shoulder and elbow joints. Significantly, very frequently noted incident to emergence from under high pressures in diving and caisson operations, as analysis of the data in the literature shows, is precisely this distribution of bends symptoms in terms of frequency of involvement of these or other joints. As regards the nature and dynamics of development of painful sensations, which were carefully analyzed on the basis of the detailed reports of examinees, as well as on the basis of self-analysis of these sensations in self-experiments by V. P. Katuntsev and myself and compared with the data in the literature on painful sensations incident to emergence from under high pressures, no differences of principle could be detected between "altitude" and "depth" bends. The well-known fact that painful symptoms incident to emergence from under high pressures are generally distinguished by their high intensity concerns the quantitative, and not the qualitative side of the matter.

Thus, the analysis of the localization and nature of altitude decompression pains undertaken by V. P. Katuntsev has shown that the latter do not differ qualitatively in any respect from /329 decompression pains that arise after a stay under increased pressure. On the basis of this cardinal result of the study undertaken by V. P. Katuntsev, we can conclude that both the internal conditions (the intimate mechanism of development of painful symptoms) and the localization of the gas bubbles that cause these symptoms, incident to the action of a rarefied

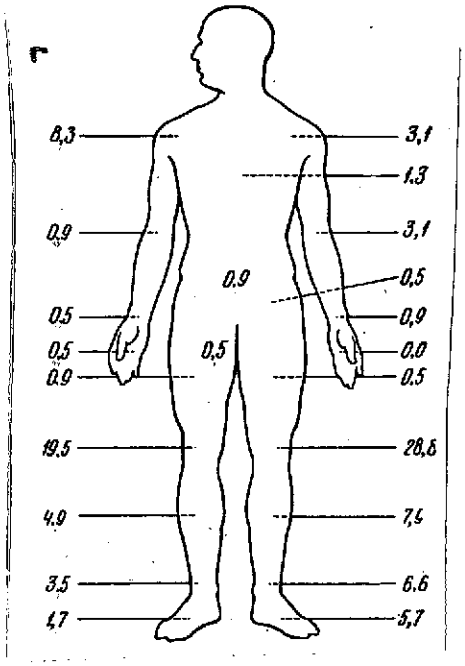

Fig. 79. Localization of painful symptoms of altitude disorders in examinees. The figures indicate the frequency with which painful decompression symptoms arise in the corresponding regions in percent of the total number of cases of decompression disorder development (192 cases).

atmosphere, as well as incident to emergence from under increased pressures, obey a common principle. This affords new general points of support for analysis of those standard regularities in the development of decompression disorders which are valid for the different concrete conditions of their appearance.

As we can see, the experimental data set forth in the present chapter do not make it possible to solve the problem of the localization and intimate mechanism of development of painful decompression symptoms. This would have been too much to expect inasmuch as this problem, as has already been noted, has been the object of attention of researchers for more than 100 years now and has remained and still remains unsolved to this day. But it can be hoped that the experimental facts presented above are not useless. They give additional grounds for consideration of this problem and for its further experimental study.

CONCLUSION

In conclusion, we must dwell on still another question of /330 principle that was raised in the very beginning of our study when we were discussing the terminology of decompression disorders. It is a question of whether it is legitimate to identify all cases of decompression disturbances with disease, just as whether it is legitimate in general to identify with disease those states of the organism differing from the norm which arise incident to the action of various extraordinary factors on it.

Before discussing the essence of this question it is necessary to make one reservation. The conclusions that will be set forth below do not belong to the category of direct and incontestible

inferences that ensue immediately from experimental data. It is possible that many of these conclusions, and perhaps even the entire question as a whole, will be considered controversial. But inasmuch as this question is of considerable scientific and practical significance (of this more will be said below) and is therefore subject to discussion, it did not seem useless here to report on the essence of that point of view which took shape in the course of the systematic investigations conducted by M. P. Brestkin and his collaborators and which is devoted to the organism's interaction with extraordinary factors. This point of view rests to a considerable extent on the experimental data presented in this book.

To identify those special states of the organism which arise under the action of extraordinary external factors with diseases has some historical justification. When people first came across such cases as hypoxemia, decompression disorders, etc, the nature of these conditions was unclear to them. Not being aware of those factors which cause the given conditions and seeing at the same time a number of manifest, and sometimes even very serious, functional disturbances involving the organism, they naturally found them similar to diseases. This gave rise to the corresponding terminology: "mountain disease", "altitude disease", "air disease", "caisson disease", etc. This terminology was /331 very much consolidated by those cases where, as a result of undergoing these effects, in particular decompression effects, people developed irreversible functional disturbances, and the casualties remained invalids for the rest of their lives. But when, owing to the indicated circumstances, the expressions enumerated above had received general recognition, they began to be used also to designate even the mildest incipient functional changes that arise incident to the effects referred to. Especially firmly established were the terms "caisson disease" and then, when its cause had been elucidated, "decompression sickness," and again people began resolutely applying these expressions to all cases of decompression-induced functional changes, including even the mildest signs of caisson phenomena developing in the organism.

As early as the Forties, M. P. Brestkin raised the question of the legitimacy of identifying the above extraordinary states of the organism with diseases in the case of altitude hypoxemia, usually called "altitude sickness." In an article by his student V. A. Skrypin in the Encyclopedic Dictionary of Military Medicine, which is devoted to this problem, it is pointed out that the wide-spread expression "altitude sickness" is essentially incorrect in that altitude hypoxemia is a special state of the organism that is absolutely different from its ordinary state, but not identical with disease.

Subsequently, many-sided research on the effect on the organism of various extraordinary factors (oxygen and carbon

dioxide insufficiency or excess, high pressures of inert gases, drops in pressure, intrapulmonary overpressure, overloads and rocking, changed temperature conditions, etc), which was being conducted in the pressure laboratory of the Military Medical Academy, showed ever more clearly that this thesis was correct not only in respect of hypoxemia, but also in respect of the overwhelming majority of extraordinary states of the organism that develop incident to its interaction with essentially changed external conditions. This idea was expressed in the textbook article "General principles of interaction between the organism and extraordinary environmental factors encountered under military conditions" [1957].[19] But it has not, in our opinion, yet been subjected to sufficient scrutiny by researchers studying the organism's activity under extraordinary conditions.

The factual material presented in this book constitutes new evidence of the correctness of this idea. Essentially it says that if the chief sign of any disease is impaired functions of the organism under ordinary (normal) external conditions, /322 then under the action of extraordinary factors its functions will be changed only insofar as external conditions are changed. In the first case, the pathologic process developing in the organism disturbs the interrelations between the organism and the environment to which it is accustomed; the sick organism is unable to counterbalance even ordinary external conditions. In the second case, the healthy organism enters into single combat with an extraordinary factor and very often, owing to its powerful defense-adaptive reactions, quite successfully counterbalances even conditions that are very different from ordinary conditions.

In the first case -- that of sickness -- many functional shifts (for example, panting and tachycardia in pulmonary or cardiac patients), of compensatory significance as they are, nevertheless emerge most of all as indices of a state of disease, as symptoms by which to pass a correct judgment on the disease. Thus, panting in a patient from heart failure emerges most of all as a symptom of disease; it does have some compensatory significance, but is only an extreme measure permitting the patient to maintain life and, what is more important, it can in no way influence the pathological process underlying the disease.

In the second case -- that of interaction of the organism with extraordinary factors (for example, oxygen insufficiency, high temperature) --these same shifts emerge most of all not as pathological symptoms, but as indices of full-fledged defense

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[19] In the book: Selected topics in military medicine, edited by M. P. Brestkin, Leningrad, 1957, pp. 18-42.

reactions that the organism marshals against the harmful effect. In this case, these reactions directly influence the cause of the functional disturbances; it reduces the effective force of the extraordinary factor on the organism. Thus, panting caused by oxygen insufficiency in the inspired air reduces the degree of hypoxemia. Under these conditions, in contrast to ordinary conditions, the absence of the above functional shifts would attest to an abnormal reaction of the organism to the given concrete conditions.

Of course, all these paradoxes refer mainly to those cases where the intensity of the acting extraordinary factors is not excessively high and where the organism, owing to its defense reactions, still manages to counterbalance the harmful effect, even at the cost of simultaneously arising functional disturbances. It is precisely these cases, by the way, that are of primary interest in practice.

But even incident to a considerable intensity of a noxious factor, when together with defense reactions, a number of serious functional disturbances arise and compensation for the extraordinary effect becomes patently insufficient, the state of the organism still must not be identified with disease. If, in such cases, the noxious factor is eliminated without delay, the /333 functions of the organism become normalized rapidly and completely. In contrast to this, the pathological process underlying any serious disease cannot be instantaneously interrupted and stopped.

All these, as it seems to us incontestible, differences between true diseases and special states of the organism that arise in connection with its interaction with extraordinary factors, in no way contradict one obvious truth: incident to a certain intensity and duration of its effect, any one of these factors can cause real and, in a number of cases, serious and irremediable disease; more exactly, it can lead to an irreversible pathological state which, as a rule, is already based on morphological damage and which can profoundly incapacitate a man. But to establish whether or not a true disease arose under the effect of an extraordinary factor usually proves to be possible only when it has been removed; as long as the extraordinary factor continues to act on the organism, it is difficult to judge whether or not in the process of this interaction, that boundary has been passed after which normalization of conditions can no longer lead to normalization of functions, and the organism proves to be damaged and becomes sick.

The experimental material presented in this book constitutes additional evidence for the above conclusions. The peculiarity of decompression effects, which consists in the fact that they are not harmful to the organism in themselves as a mechanical factor, but as the cause of the gas bubbles that arise inside the organism, gives no grounds for differentiating as a matter of

principle these effects from all other extraordinary effects of a changed gaseous medium. To tell the truth, in no case does an external effect tell on the organism's activity except in virtue of inducing inside the organism changes of one sort of another that are the direct cause of functional shifts. Incident to decompression disturbances, the directly effective factor is the gas bubbles arising inside the organism, the formation of which is due to a change in external conditions -- an extraordinary decompression effect.

Experiments show that to this extraordinary factor -- gas bubbles forming, in particular, in the blood -- the organism at first responds with pronounced defense reactions. When decompression effects are not distinguished by any particular intensity, their consequence reduce almost exclusively to development of defense reactions -- increase in pulmonary ventilation and intensification of heart action; on account of these reactions, the extraordinary effect is counterbalanced by the organism, the gas bubbles that have arisen are soon liquidated and the formation of new ones comes to an end -- the extraordinary factor ceases to have an effect, and the functions of the organism become normalized. These cases, which are undoubtedly the ones /334 most frequently encountered in practice, must not be classed with diseases. Figuratively speaking, they constitute a short battle that the healthy organism puts up with a suddenly attacking, but comparatively weak, enemy, a battle that ends in rapid and complete victory.

Adding to the described cases such phenomena as itching of the skin, marbling and burning of the skin does not fundamentally change the picture. These phenomena, which always arise only against a background of general adaptive reactions of the respiratory and cardiovascular systems that have already been set in motion, reflect not only the unfavorable consequences of the local effect of gas bubbles, but also the development, in response to this, of local defense reactions; as a rule, they practically do not disturb the organism's efficiency and even disappear rather rapidly without any therapeutic measures. But it is customary to regard these phenomena as specific symptoms of caisson "disease;" on their basis, researchers in all categoricalness speak of the onset of decompression "sickness" taking a mild course. But again the matter practically reduces to a short-lived and victorious battle of the organism with extraordinary factors.

As regards decompression disturbances of the bends type and other, more serious, decompression disorders, externally and formally, they can indisputably be subsumed under the rubric of diseases. But even these decompression disorders, up to a certain time after their onset, differ from diseases. So long as there is no "breakdown," to use I. I. Pavlov's descriptive expression, no disease as such yet exists, but only different more or less serious proximate consequences of the extraordinary

factor acting in the internal environment, consequences that arise against the background of the organism's relentless battle with the harmful effect. Practice and experience leave no doubt about one thing: timely application of recompression liquidates with amazing rapidity often even extremely serious functional disturbances and normalizes the organism's functions.

Consequently, in all cases where the matter has not yet progressed to a certain boundary, it suffices to eliminate the harmful factor for the organism's functions to be rapidly normalized.

As a rule, with diseases the case is different as a matter of principle. In most cases, the initial etiological factor that caused the disease, and the host of conditions under which it manifested its pathogenic effect, in the course of development of the disease lose their determinative significance and very often completely cease to play any role at all. "Once a disease has arisen," as is pointed out by I. R. Petrov and Z. K. Kulagin [1966], "it develops further in conformity with its own internal laws as a result of different phenomena connected with etiological relations."[20] To stop this internally caused development of a disease instantaneously and return the organism to normality proves to be impossible. /325

Finally, there is still another extremely important circumstance that distinguishes diseases as a matter of principle from those states of the organism which arise under the effect of extraordinary external factors. This circumstance comes to light most strikingly on the example of decompression effects. The point at issue is that, on the one hand, the consequences of recurrent diseases, be they ever so mild (for example, catarrh of the upper respiratory tract, bronchitis, angina, etc) and, on the other, the consequences of recurrent, not excessively intense, extraordinary factors.

The inevitable consequence of frequently recurrent diseases is a reduction of the organism's resistance to the different pathogenic effects in its internal environment, a depletion of its reserve stores of physiological resources, a restriction of its adaptability to surrounding conditions; in a word, a weakening of health.

The regular consequence of recurrent, not excessively intense, extraordinary factors, as has been shown in the present study on the example of decompression disorders, is an increase

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[20] Handbook of pathological physiology, Vol. 1, Meditsina, Moscow, 1966, pp. 87-88.

in the organism's resistance to the given factor and, consequently, an increase in its physiological reserves and an expansion of its adaptive resources. There is every reason to suppose that the effect of such conditioning to the action of a number of extraordinary factors, provided their intensity in the course of conditioning was kept within proper limits, is not confined only to an increase in resistance to their influence, but is accompanied by an increase in the total resistance and efficiency of the organism; in a word, a strengthening of health.

Strictly speaking, this conclusion ensues of itself from the practice of different special occupations involving the effect on the organism of extraordinary external factors and, primarily, the practice of diving, aviation and, especially, astronautics. The specificity of all complex and prolonged preparation of divers, fliers, and, especially, astronauts consists essentially in conditioning the organism to the action of various extraordinary factors and to the performance of required work under changed external conditions. There is no need to prove that such preparation not only does not shatter the health of fliers and astronauts, but, on the contrary, to a considerable extent strengthens it. From these well-known and indisputable facts alone, it becomes clear that it would be a big mistake to equate with diseases the different cases of the organism's interaction with extraordinary factors. /336

It follows from the foregoing that the term "disease" or "sickness" (altitude disease, air sickness, caisson disease, decompression sickness), which is employed to designate the frequency of occurrence of specific reactions of one kind or another incident to the action of different factors -- in particular, decompression -- must also be discarded.

It is necessary to stress, however, that the differences of principle between diseases and those states of the organism which arise incident to its interaction with extraordinary factors give no grounds to conclude that these states, inasmuch as they are not diseases, present no grave danger to the organism. Such a conclusion, besides being essentially incorrect, might bring in its wake much practical harm and occasion unfortunate cases and human victims. There can be no doubt that any one of these extraordinary factors, even such as appear comparatively harmless at first glance, can, under certain conditions, become pathogenic, and many powerful factors threaten the organism with the loss not only of health, but also of life.

Decompression effects, which have been the subject of our investigations, are one of the most serious and dangerous effects of a changed gaseous medium. Their distinctive feature resides in the fact that the factor -- gas bubbles in the

organism's internal environment -- act latently and are not subject to any calculation or determination. Of its onset and the beginning of its effect and of whether it weakens or intensifies and becomes threatening, we can judge only by the organism's externally noticeable reactions. The organism's powerful adaptive resources in respect of the given factor aggravates its insidiousness: against a background of apparently complete well-being, which has until then been ensured by full-fledged compensation, very serious and irremediable functional disturbances may suddenly develop.

In connection with all of the foregoing, it might be asked why it should be particularly necessary to pay so much attention to the differences between diseases and cases of the organism's interaction with extraordinary external factors and whether this question is not of an abstract, academic nature. It is our unshakeable conviction that this question is of paramount practical and theoretical importance and, consequently, merits the most detailed analysis and requires a resolute and unqualified solution.

The scientific and practical significance of this question resides in the fact that one or another of its solutions will determine the approach of researchers to the treatment of the different problems connected with man's activity under extraordinary conditions and, consequently, the nature, results and progress of this treatment. At the present time, when a great number of physicians of the most diverse specialties are being drawn to the problem of man's activity under extraordinary or, as it has become customary to say, extremal conditions in connection with the task of developing the depth of the sea and, especially, in connection with the task of exploring space, it is absolutely necessary to determine the general principles of approach to evaluation of the state of the organism placed under changed conditions.

If the researcher approaches the organism placed under extraordinary and often very difficult conditions as if it were a sick organism, he will always be hounded by the danger of incorrect evaluation of phenomena: to him, the different functional shifts will look most of all like symptoms of disease; the adaptive reactions of individual physiological systems, and, above all, the adaptive reactions of the organism as a whole, will remain outside his field of vision; the entire mechanism of the organism's interaction with extraordinary conditions will appear in an incomplete and, perhaps, even a distorted light. But in evaluating the organism's interaction with extraordinary factors as a process of battle with their unfavorable effect, the researcher will rapidly see what is of greatest practical and theoretical importance: the mechanisms of man's possible adaptation to activity under changed conditions. In so doing, he will not risk omitting from his observations different

functional disorders and disturbances -- the latter will always get a hearing and will not pass unnoticed.

Orientation on the adaptive resources of the organism in respect of extraordinary factors, besides meeting the requirements of practice, is justified from the theoretical point of view. It is by precisely such an orientation that special, as well as general, physiology will be enriched with the results of studying the organism's activity under extraordinary conditions and elucidating its adaptive resources.

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